THE PLANT DISEASE REPORTER

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Division of Mycology and Disease Survey

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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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IN THIS ISSUE

John P. Hollis sets forth a hypothesis of the origin of plant diseases from nonreactive associations of microorganisms and higher plants, page 219.

Systox controlled root-knot nematodes when used as a soil drench in certain concentrations in experiments reported by J. N. Sasser, page 228, but the possibility of toxic residues makes further investigation necessary before the chemical can be recommended for this purpose.

Temporary soil sterilization did not provide a basis for estimating losses from root and crown rots of wheat and barley, in experiments reported by G. W. Bruehl, page 234.

Bacterial wilt of corn will probably be more destructive in Illinois this year than last, according to the forecast sent by G. H. Boewe, page 238.

D. M. McLean reports occurrence of the yellow-net virus disease in table beet seed plants in northwestern Washington, page 241; the disease apparently is caused by the same virus that causes yellow-net of sugar beets in California.

The chrysanthemum stunt virus possesses exceptional heat tolerance, according to Philip Brierley, reporting on various properties of the virus, page 243.

C. E. Horner reports diseases occurring on peppermint in Oregon in 1951, page 245.

Unusual hosts for two southwestern mistletoes are reported by Frank G. Hawksworth, page 246.

Observations reported by Paul R. Miller and G. Flippo Gravatt, page 247, show that the sweetgum blight first reported in a limited area in Maryland in 1948 has spread over much additional territory.

Unusual records, page 253, contain reports on discovery of the potato golden nematode in Peru, by C. Bazán de Segura; presence of root-lesion nematodes in Christmas rose imported from Holland, by C. G. Anderson and H. J. Jensen; finding of oospores of Albugo candida in horseradish leaves in Illinois, by R. M. Takeshita et al.; and a note on the root-rot disease-complex in tobacco plant beds in Virginia and North Carolina, by Wilbert A. Jenkins.

A correction, page 254.

Review of "Disease in Plants", page 255.

April weather, page 257.

ON THE ORIGIN OF DISEASES IN PLANTS

John P. Hollis

A search of literature on bacteria-higher plant relations revealed that the presence of bacteria in the vascularized portions of healthy potato tissue (14, 29) was generally unexpected and that there was no theory of microorganism-higher plant relations admitting such a possibility. A survey of this entire field of relations indicated that knowledge had been segregated into compartments bearing such names as mycotrophy, root diseases, rhizobia-legume symbiosis, rhizosphere effect, mycodomatia, epiphytic flora of plants, mycorrhiza, bacteriorrhiza and diseases of various crops.

In the absence of unifying theories of relation or origin, progress in alien compartments has been largely ignored by all workers. Existent theories are limited conceptually and are shot through with teleological reasoning and time-ingrained rationalism. For example, Kelley (20) states that of 118 papers dealing with observations of the mycotrophic conditions in plants, in which the writers expressed a definite opinion about the endophytic relationship, only 14 voted for parasitism. Whatever the correct relation may have been in the majority of cases it is obvious that mycotrophy at this time had hardly attained the status of a science!

During the past 75 years sufficient knowledge of microorganism-higher plant relations has accumulated to indicate that nonreactive, symptomless, relations comprise, by far, the bulk of all relations and that reactive or symptom-producing relations are, comparatively, of rare occurrence. As a reflection of this fact, I would like to advance the hypothesis that reactive relations have evolved from nonreactive relations (Figure 1). If any question exists as to whether a relation is reactive or nonreactive it should, in the absence of adequate biochemical criteria, be considered as the latter. Such a hypothesis should discourage teleological speculation because there is implicit in it the idea that the non-reactive relation contains some elements of chance. Further it should be unnecessary to rationalize observations and to "call" the relation either beneficial or harmful to the higher plant without substantial evidence along biochemical or physiological lines.

The present approach to the problem of the origin of diseases in plants draws heavily on the time-honored idea that disease-producing organisms arise as a result of mutation and selection among saprophytes. The phraseology of this paper broadens the concept to include all categories of microorganism-higher plant relations.

It has been argued that the wealth of information on rust fungi and on viruses should be considered in any treatment of the origin of disease (27). The development of such obligate relations might be associated with the loss by the microbial or higher-plant component of enzymes necessary to synthesize cell material as has been shown for the parasitic component of animal diseases (7). Intensive treatment of these data would necessitate, however, equal consideration of a large group of diseases caused by "low-grade" pathogens such as Rhizoctonia, Sclerotium, Phymatotrichum and Streptomyces. These organisms appear to be nutritionally independent of the suscept, surviving vegetatively in its absence. They produce necrotic lesions suggestive of toxins and attack a wide variety of plants. Additionally, there is sufficient evidence in general microbiological literature to suggest a toxin theory of disease, which may find its future support resting largely on studies of these disorders.

In spite of these advances there is a notable lack of experimental data concerning the manner in which microorganisms mutate or adapt and become pathogenic. Consequently, it would appear that neither a separate nor unified comprehensive treatment of these two groups of diseases from the standpoint of origin, should be attempted at present. The treatment in this paper is necessarily superficial and taxonomic. It is primarily a review, not comprehensive, but illustrative of the tenets, corollaries and consequences of the hypothesis presented.

OUTLINE OF MICROORGANISM - HIGHER PLANT RELATIONS

In Figure 1 is a diagram of all known microorganism-higher plant relations. It may be noted that each reactive complex possesses a nonreactive counterpart; indeed the two may be visualized in "natural equilibrium" whereby one relation may be transformed into another. The current hypothesis is based largely on the fact that the equilibrium of all relations in nature is shifted far to the left, i. e., toward the nonreactive side. For reasons which will become apparent the diagram departs from an idealized scheme of relations in two respects: (a) irreversability of certain mycorrhizal affinities and (b) apparent unrelatedness of the bacteriorrhiza phenomena with reactive counterparts involving cortical tissues.

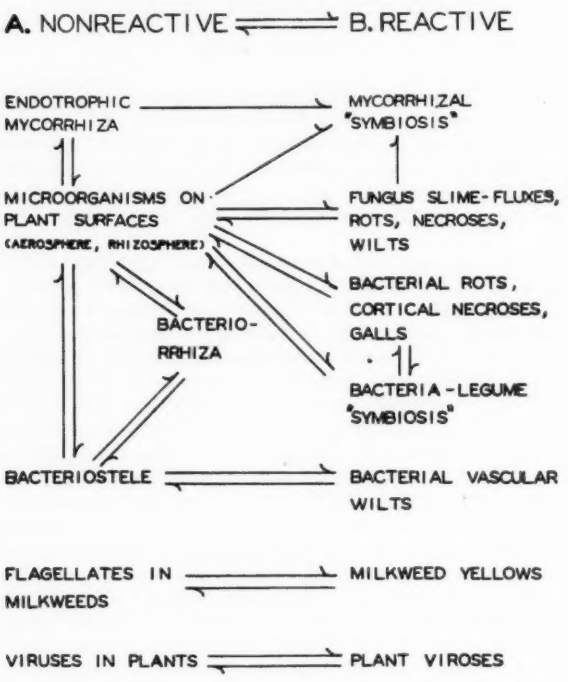

FIGURE 1. Diagram of microorganism-higher plant relations illustrating the hypothesis that reactive relations originate from nonreactive relations.

There are many examples of both nonreactive and reactive mycorrhizas (fungus-root associations). A nonreactive condition is indicated by the results of Armstrong and Armstrong (2) who recorded the presence of elegans-type Fusaria in the roots of nonsusceptible hosts, and reactiveness in the extensive and painstaking observations of Jones (18) of the phycomycete-like fungus in the cortical tissue of legumes and other herbaceous types. Jones found that the fungus projected haustoria into cells causing them to become discolored, rendering the entire root yellow. This discoloration coupled with slow growth of plants at the time of maximum root invasion indicated that the effect was probably detrimental to the plant.

Reactive relations may be distinguished from nonreactive relations on a local or point of association basis or on a general or humoral basis. For purposes of simplicity, i.e., in order to avoid compensating effects, only the local reaction will be considered. Reactiveness in the ectotrophic type of mycorrhiza is characterized most commonly by the coralloid type of root growth; while in the endophytic type it is exemplified by the results of Jones described previously. Actually most endophytic relations are symptomless and should be considered nonreactive in spite of the complication introduced by the intracellular digestion of mycelium.

The foregoing criteria indicate that many mycorrhizal relations could be considered reactive and the equilibrium then shifted to the right in Figure 1. Studies of the fungus flora of roots are sufficiently numerous and comprehensive, however, to suggest a common occurrence of nonreactive relations. Katznelson, Lochhead and Timonin (19) in recent review pointed out limited evidence in support of a true fungus rhizosphere effect exerted by the roots of plants studied and modified by soil factors such as hydrion concentration. The present writer's concept of nonreactive mycorrhiza envisions association of fungi with the roots of plants in terms only of proximity. Indeed, evidence is available, Kelley (20), to show that mycorrhizal fungi are those commonly found in soil.

As used in the literature the term mycorrhiza denotes a fungus-root association detected by either macroscopic or microscopic symptoms and signs. In this classical sense a common

type is the endophytic association of phycomycete-like fungi with the roots of flowering plants and cryptogams. Hypothetical linkage between these endophytic associations and diseases caused by members of the Peronosporaceae is suggested by the resemblances between the fungal components and by the nature of the association, generally confined in both instances to cortical tissue. Rayner (28) considered the type of infection by mycelium bearing vesicles and arbuscules and the presence of intracellular digestion favored the conclusion that the endophytes of many vascular plants belong to a single group. The fact that no endophyte of this type had yet been isolated was taken as an indication of specialization to the symbiotic habit and may be cited here as support for the idea of irreversability outlined in Figure 1.

The number of fungus diseases far exceeds the number of proven cases of beneficial relations in spite of numerous attempts to establish the occurrence of the latter where doubt has existed. Nitrogen fixation as a factor, leading to beneficial effects, has been eliminated from serious consideration in nonreactive types of relations, although curiously enough, several instances of its occurrence in highly obligate reactive relations have been confirmed, Foster (8). Actually there is no clearcut region between reactive relations harmful or beneficial to the participants. As Gäumann (9) states, "In these borderline cases where the host derives a more vital advantage than the parasite from the parasitic relation, discussions on the 'significance of the mycorrhizal formation' really deteriorate into arguments about words. What is pertinent to our study of parasitology is that as in the root nodules of the Leguminosae, so in the mycorrhiza, plasmatic defense reactions regulate the cohabitation of parasite and host by their crippling, localizing and eliminating effects." In the light of this statement the irreversability of mycorrhizal "symbiosis" with fungus diseases (necroses and rots, etc.), indicated by the arrow in Figure 1, represents merely an "adjusted" relation permitting continuity of association. The "symbiotic" relation is of higher order in point of time necessary for its evolution by mutation and selection.

The preponderance of nonreactive over reactive associations in bacteria-higher plant relations is well established by records of the ubiquitous occurrence of bacteria on plant surfaces, Smith (30). The rhizosphere relation is identified by the presence of typical bacteria on plant roots while the bacteriorrhiza relation is differentiated by the diffusion of bacteria into the root cortex. These relations are nonreactive in that their higher plant components do not exhibit symptoms. Reactiveness, however, is manifested by the bacteria which increase in numbers. The bacteriostele relation, identified by the presence of bacteria in vascular elements, appears, on the basis of available evidence, to be nonreactive in both components. These three relations have been detected in numerous plant species. All plants examined by suitable experimental methods have been found to posses epiphytic bacteria and to exert a rhizosphere effect. If the search were extended, doubtless, the bacteriorrhiza and bacteriostele relations would be found to occur commonly in many species. These facts coupled with the small percent of plants, visibly diseased as a result of similar associations, indicate the natural equilibrium in bacteria-higher plant relations to be far to the left.

In Figure 1 the bacteriorrhiza relation is placed between surface microorganisms on the roots of plants (the rhizosphere and aerosphere relations) and the bacteriostele relation (bacteria in vascular elements of plants). This relation was characterized by Perotti and his associates (24) who found formations of bacteria in the roots of over 75 percent of a large number of plant species. The bacteria were largely intercellular in the cortex but occasionally were found to be intracellular. While in general the various relations were apparently nonreactive, the authors reasoned there was a symbiotic effect of mutual benefit as a result of the presence of the bacteria, which had been shown to possess marked fermentative powers in pure culture. Like the mycorrhizal fungi the bacteria were found confined primarily to cortical tissue. Isakova (16) has confirmed the existence and essential features of the bacteriorrhiza association. The apparent nonrelatedness of this association with bacterial cortical necroses, galls and legume "symbiosis" stems from wide differences in the types of bacteria involved.

The frequency and higher plant range of the aerosphere and rhizosphere relations is evidence of the preponderance of this type of relation over possible reactive counterparts as postulated in Figure 1. The rhizobium-legume association is of limited occurrence and cortical necroses, taken over the entire plant kingdom, although numerous, are rare in a comparative sense. Valleau, Johnson and Diachun (35) demonstrated that certain pathogenic leafspot bacteria are associated with the roots of nonsuscepts. Such relations, scarce at the moment, are perhaps more common than present knowledge indicates. Since there is no evidence of mutualism in the bacteria-plant surface relations it should strengthen possible ties with the reactive rhizobia-legume "symbiosis" to point out that Gäumann (9) and Allen and Allen (1) consider the rhizobia to be facultative parasites, a position entirely tenable, when the reaction is viewed only from the

standpoint of cells invaded, excluding possible compensatory effects on the plant as a whole.

The bacteriostele relation is of special interest because of its evident nonreactiveness. Occurrence of small numbers of bacteria in a high percent of isolations and in all plant species critically examined suggests linkage with bacterial vascular wilts and a natural equilibrium far toward the nonreactive side (14, 29). Several modes of entry of bacteria into the vascular elements have been proposed (14, 34) but evidence in support of these hypotheses is entirely circumstantial. Consequently it seemed reasonable to place the bacteriorrhiza relation between the rhizosphere and bacteriostele relations in conformance with gradation of depth of invasion of the plant roots. Such mass action as is exerted by formations of bacteria upon the surface and in the cortical tissues of the roots should create a situation conducive to passive invasion of vascular elements along all possible avenues of entry.

Nonreactive associations exist in virus-vascular plant relations. The first account of such a relation was probably that of Johnson (17). The discovery of a nonreactive flagellate-milkweed relation was detailed by Holmes (15) where previously only reactive situations had been thought to occur, Lafont (21). Nothing is known of the relative proportions of reactive and nonreactive relations in these two groups.

AFFINITIES AMONG BACTERIA IN THE DIFFERENT ASSOCIATIONS

The outline (Figure 1) is based on the probability that affinities exist between apparently similar types of associations. While chance association may or may not furnish the requisite conditions for selection in nonreactive relations there is, in the reactive types, immediate selection and the conditions for continuous modification by mutation and selection, which in the fungi eventually results in highly obligate combinations such as stem rust of cereals. Similar obligate relations derived along slightly divergent lines are homologous and bear the affinities of common origin and yet cannot be transformed, one into the other, because of differences which are precisely those of the particular component endophytes and higher plants. The microorganisms of these relations are in most instances not amenable to isolation and study alone because they cannot be cultured apart from their associants. Consequently the fungi cannot be compared physiologically over the entire spectrum of their relations with higher plants.

The bacteria, however, exhibit a variety of facultative associations with higher plants and the wealth of their morphological and physiological types makes it likely that a comparison of affinities will serve to strengthen the present scheme of relations.

Epiphytic bacteria or those from the aerial parts of plants (aerosphere) were found by Düggeli (6), in a comprehensive survey, to consist of a few abundant types, three of which must have been species of Pseudomonas (3). Less numerous were several well known members of the genus Bacillus. In reports of isolation of miscellaneous species from plant surfaces gram negative rods and gram positive cocci are common (3).

While knowledge of the aerosphere flora is limited, that of the rhizosphere is well known. In a recent review, Katznelson, Lochhead and Timonin (19) stated, "The weight of evidence suggests that gram-negative, non-sporeforming bacteria (Radiobacter types, fluorescent and "dye-tolerant" forms) multiply extensively in the rhizosphere of a wide variety of plants, whereas sporeforming bacteria do not. The latter can develop on roots and occur in greater abundance on those of certain plants (oats, radishes) than on others (barley, corn, beans, lettuce)." The Lochhead group (19) has found there is a relative increase in the rhizosphere of organisms with simple requirements and of those requiring amino acids for maximum growth and a relative suppression of bacteria dependent upon the more complex nutrients provided in soil extract. Corynebacteria are found in the rhizosphere of plants but are, Lochhead (22), relatively less abundant there than in soil apart from the roots.

The limited descriptions of Perotti and his associates (24, 25, 26, 36) indicate the bacteria of the bacteriorrhiza relation to be gram positive, motile, non-spore-forming rods and, therefore, morphologically unrelated to predominant forms of all known reactive relations. In light of our limited knowledge of bacteriostele bacteria there may be some affinities with these forms as indicated in Figure 1.

Bacteriostele types resemble those commonly found in the rhizosphere to a remarkable degree (14). Sanford (29) reported isolation of the common rhizosphere form Bacterium radiobacter from the interior of potato stems. Bacillus megatherium DeBary has been reported of some prevalance in both aerosphere (6) and rhizosphere of numerous plants (4).

With the exception of those diseases produced as a result of association of the gram positive Corynebacteria with higher plants, all reactive associations are with the participation of gram negative forms. There is then, excellent correspondence among aerosphere, rhizosphere,

and bacteriostele forms and pathogens inducing necroses and wilts and the rhizobia. This agreement extends to both morphological and physiological characteristics. For example, the wilt producing bacteria are physiologically rather homogeneous in spite of ranging over five genera of classification types and resemble rhizosphere forms in general level of nutritional competence, (Table 1 from Hildebrandt (13)). The studies of Starr (31, 32, 33) on the nutrition of Agrobacterium, Xanthomonas and Corynebacterium lend support to these conclusions.

Table 1. Comparisons between characters of various wilt inducing bacteria. Data from Hildebrandt (13).

Organism	Aerobe	Gelatin liquefaction	Nitrate reduction	Chromogenesis	Carbon source reaction				Gram	Capsules	
					Acid, glucose	Acid, lactose	Acid, sucrose	Acid, glycerol			
Corynebacterium insidiosum	+	+	0	Yellow	+	+	+	+	+		
(Xanthomonas phaseoli)	+	+	0	Yellow	+	+	+	+	0	m	+
(Pseudomonas phaseolicola)	+	+	0	White	+		+	+	0		
(Xanthomonas campestris)	+	+	0	Yellow	+	+	+	+	0		+
Erwinia tracheiphila	+	0	0	White	+	0	+	+	0		
(Erwinia amylovora)	+	+	0	White	+	+			0		0
Bacterium stewartii	+	0	0	Yellow	+		+	+	0		+
Xanthomonas vascularum	+	+	0	Yellow					+0		
Pseudomonas solanacearum	+	+?	+	Brown	+		+	+	0		
Corynebacterium michiganense	+	+	0	Yellow	+	Tr	+	Tr	+		+

+ = Positive; 0 = Negative; Tr = Trace

Table 2 constructed from data assembled by Dowson (5) shows a breakdown of bacterial diseases into three categories. Of 62 species, 45 induce diseases of the parenchymal tissues; 10 attack both parenchymal and stelar tissues, i.e., are systemic; and 7 attack the stelar tissues. Of interest is the relation between number of diseases and depth of penetration into the plant. That individual species must be capable of inhabiting the aerosphere or the rhizosphere before mutation and selection can bring about reactive conditions is implied in preference to the reverse view, that intrinsic pathogenic capabilities exist due to mutation in absence of the plant followed by selection after chance association. The fact that rhizosphere and pathogenic forms are so closely related morphologically and physiologically furnishes support also for the idea that the plant "selects" certain types of bacteria and that it is with these types that the majority of reactive combinations arise. Nonrhizosphere forms such as Streptomyces scabies (Thaxt.) Waksman and Henrici possibly become abundant and reactive on the peridermal surfaces of tubers and thickened roots because the suppressive or competitive influences of the normal rhizosphere surfaces are absent in these organs.

Table 2. Number of bacterial plant pathogens invading different parts of the plant. (Data compiled from Dowson 5).

Genus	Number of species invading different parts of the plant			Totals
	Parenchyma	Parenchyma-stele	Stele	
Pseudomonas	25	3	0	28
Bacterium	9	0	2	11
Corynebacterium	1	4	1	6
Xanthomonas	10	3	4	17
Totals	45	10	7	62

MECHANISM OF ORIGIN OF REACTIVE RELATIONS

The hypothesis that nonreactive associations change to reactive ones presupposes mechanisms which regulate rates of origin and occurrence of new reactive combinations. By analogy with theories of microbial resistance to drugs it can be hypothesized that new diseases arise as a result of (a) one or more spontaneous mutations in one or both associates followed by biological selection for a reactive condition or (b) chemical adaptation and biological selection from the general population. This type of adaptation is produced as the result of a reaction by a microorganism to the presence of a specific substrate not commonly encountered. The substrate exerts some influence on the developing enzyme proteins, resulting in the production of an enzyme able to absorb and decompose it. Since the ability to adapt in this way is possessed by a population of microorganisms as a whole, the mechanism of chemical adaptation, if the only one operative in the evolvement of new reactive combinations, would be expected to give rise to a much higher proportion of these relations than now exist. Indeed -- the spontaneous mutation and biological selection theory is supported by the fact that there exists a great preponderance of nonreactive associations in nature. The relatively small numbers of reactive combinations in different types of associations are explicable in the light of selection from small mutant populations.

While nonreactive relations are symptomless there is in the aerosphere, rhizosphere and bacteriostele relations a strong biological selection for certain microbial components. Elements of chance are involved but these relations might arise primarily as a result of chemotrophic responses. Mutants for pathogenicity situated at the loci of association would be readily selected for the reactive condition while the bulk of mutants for pathogenicity in chance or nonassociating microorganisms would be lost. The sequence of events leading to a reactive condition might be chance association of the microorganism and higher plant followed by biological selection resulting in a symptomless (nonreactive) relation, then spontaneous mutation in one or both associants followed by biological selection for a symptom-producing (reactive) relation.

The rate of origin of new reactive combinations by mutation and selection is a problem of both theoretical and practical importance to plant pathologists but to date has remained untouched. Practical considerations are paramount where attempts are being made to establish new associations of crop plants.

If spontaneous mutation is independent of the substrate (23), then change to the pathogenic condition by soil-inhabiting saprophytes as well as by rhizosphere forms, and forms currently inducing diseases of low specificity, should result in the presence in new areas of an excessive number of mutant pathogenic types which are then selected by the introduced suscepts. Under these conditions the initial rate of appearance of new diseases in a crop would depend almost entirely upon the number of labile mutant forms accumulated and the number of plants, and would not be related to the mutation rates of the organisms as such until the excess disease-producing potential resulting from "stored mutants" has been exhausted. The first year prevalence of diseases in crops introduced into new areas is more suggestive of this line of reasoning than possible disease introduction with the suscept. Examples of such diseases are potato scab, Goss (11) and potato brown rot, Gratz (12).

The mutation rate of a microorganism (equation 1) is a function of the proportion of mutant cells in terms of the total cells and the number of generations required to produce such a ratio. The mutation rate is usually calculated as number of mutants per cell per fission cycle. Depicting the factors regulating the number of reactive types of diseases in a crop is equation 2. The factor "total plants" may involve summation of totals for several strains, and "time" the number of generations of plants. Final units should be in reactive types (distinct diseases) per plant per generation. If the suscept variability is ruled out by clonal propagation or seed of genetic purity, then the equation need contain no factor for number of suscept types. The exponent (x) is used on the assumption of the plants being coated by a monocellular film of microorganisms from which biological selection for pathogenicity results in an increase in the third dimension (depth of film).

$$(1)\quad \text{M. R.} = \frac{\text{mutant cells} \times \text{generation time}}{\text{total cells} \times \text{time}}$$

$$(2)\quad \text{Number Reactive Types} = \frac{(\text{mutant cells})^{x}}{(\text{total cells})} \times \text{total plants} \times \text{time}$$

Obligate relations such as stem rust of cereals present a different picture. Here the number of reactive combinations will be dependent not only on the size of the crop but also on the intensity of infection. Mutation results in a modification of existing infection types and not the acquisition of basic pathogenic capabilities. Considering the barberry it is obvious that the number of mutant forms (races) extant is also a factor in determining numbers of new reaction types by recombination processes.

Equations 1 and 2 posses a common factor (mutant cells/ total cells) which permits their simultaneous solution. Equation 2 will be useful in serving as a theoretical check on equation 1, as a test of its validity. While, at the moment, these equations may be of little value in determining, say, after 25 years the total number of Fusarium root diseases expected in 50,000 acres of newly opened irrigation project devoted to the growing of potatoes in a western State; they do emphasize the need for theoretical tools whereby the foundations of knowledge pertaining to the origin of certain diseases in plants can be laid. Such preliminary determinations as may be made from direct experiment or compilation of data can be of aid in testing their validity. One critical point concerns the magnitude of the selection factor for the right member of equation 2. Such a factor represented here by the exponent X might be indicated by discrepancy in the proportions of mutant cells between equations 1 and 2. Another unknown variable is the number of mutations required to convert an organism from the saprophytic to the pathogenic condition. An interesting case of a probable one-step transition involving the tomato wilt Fusarium has been reported by Gerdemann and Finley (10).

It may be argued that a determination of mutant cells/ total cells constitutes counting the number of diseases present. This is not always true. Such information may be obtained by isolation of pathogenic forms from any source whatever and proving pathogenicity in terms of the desired crop under all expected environmental and edaphic conditions consistent with the time factor chosen. The disease may not be expressed in the field when the prediction, as to number of diseases expected, is made.

In virus diseases the mutation rate can be estimated in terms of modification of existing disease. However, such diseases cannot at this time be used to test or explain situations in pathogenically-induced diseases as a whole and are irrelevant to the present discussion.

The arguments presented here are purely speculative but they serve as a reminder that the advancement of knowledge involves both the collection of facts and the building of generalizations.

SUMMARY

1. A composite treatment of microorganism-higher plant relations shows that nonreactive relations are more abundant in nature than reactive (symptom-producing) relations.

2. A hypothesis of origin of disease from nonreactive associations of microorganisms and higher plants is elucidated.

3. A scheme suggesting probable interrelations among different associations is presented and the data assembled are discussed on the basis of affinities among the different groups of microorganisms.

4. The reactive condition probably originates in most cases from a nonreactive association

by spontaneous mutation and biological selection.

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DEPARTMENT OF BOTANY, UNIVERSITY OF MISSOURI, COLUMBIA

STUDIES ON THE CONTROL OF ROOT-KNOT NEMATODES (MELOIDOGYNE SPP.) WITH SYSTOX SPRAY (E-1059), AN ORGANIC PHOSPHATE INSECTICIDE

J. N. Sasser

In an earlier paper (3) Systox[1] was reported as having controlled root-knot nematodes when used as a soil drench. Concentrations of Systox used provided phytotoxic, but plants which did grow showed no signs of root-knot infection while the controls were severely galled. The present work was undertaken in the hope of finding a non-phytotoxic concentration of Systox which would control the root-knot nematodes when applied as a drench to infested soil.

Experiments with Tomatoes

Tomato seedlings were planted in five-inch pots of soil heavily infested with the root-knot nematode (Meloidogyne incognita var. acrita). The soil was then drenched with 300 ml. of Systox in concentrations of .005, .01, .02, .03, .05, .1, .5 and 1.0%. All dilutions were made with tap water. Each treatment was replicated five times and controls were included which were not drenched with the chemical. The plants grew for approximately 45 days in the greenhouse after which time the soil was removed from the roots by washing. The total number of galls and the number of galls containing females having egg masses, in a 5-gram root sample, were counted with the aid of a dissecting microscope and are shown in Table 1.

Table 1. Infection and reproduction of Meloidogyne incognita var. acrita on tomato seedlings grown in soil drenched with different concentrations of Systox.

Systox concentration (%)	Number plants of 5 surviving tests	Number galls per 5-gm root sample	Percent of galls containing egg masses	Root-knot index*
.005	3	48	46	4
.01	4	71	21	4
.02	5	63	36	4
.03	5	50	18	3.5
.05	4	21	33	3.0
.1	4	6	50	1.0
.5	1	0	0	---
1.0	1	0	0	---
CK	3	105	59	4

* Root knot index of cucumber seedlings subsequently grown in soil inoculated with 5 grams of infected roots from each of the treatments shown in the left hand column.

To test the viability of those egg masses produced on plants which had grown in the treated and in the untreated soil, 5-gram root samples from plants of each treatment were used to inoculate pots of soil, which were then seeded with cucumbers to serve as indicator plants. The cucumber seedlings grew for approximately 45 days, when the soil was removed from the roots by washing. The relative amount of infection is indicated by the root-knot index (4) shown in the last column of Table 1.

The results shown in Table 1 indicate that all the drench treatments with Systox reduced the

[1] Supplied by Geary Chemical Corporation, New York. Systox is principally the diethoxythiophosphoric acid ester of 2-ethyl mercaptoethanol. It is a light brown, somewhat viscous liquid and forms colloidal solution in water. It is closely related chemically to octamethyl pyrophosphoramide and is similar in its action, but has a fumigating action which octamethyl does not have.

amount of galling and of reproduction of root-knot nematodes on tomato seedlings below that of the control plants. Concentrations of .5% and above proved to be highly phytotoxic with only one of the original five tomato plants surviving the test. The .1% concentration greatly reduced the amount of root knot with little or no phytotoxicity. Apparently the Systox did not affect the viability of the egg masses produced on the treated plants as indicated by the root-knot index of cucumber seedlings used as indicator plants in the viability test. The low root-knot index for the .1% treatment was probably due to the small number of galls and egg masses on the root sample used for inoculum, rather than upon any effect caused by the chemical.

Experiments with Cucumbers

A. Five-inch pots of soil heavily infested with root-knot nematodes were drenched with 300 cc of the following concentrations of Systox: .05, .06, .07, .08, .09, .1, .2, .3, .4, and .5%. Each treatment was replicated five times and controls were included which were not drenched with the chemical. After one week, the soil in the top inch of the pots was loosened and cucumber seed planted. Six days after seeding and every four days thereafter for 22 days, growth was recorded by measuring the height of the seedlings from the soil line to the base of the terminal leaf. Lengths and breadths of leaves were measured at 14, 18, and 22 days after seeding. Length of each leaf was multiplied by each breadth to obtain the leaf area index. The growth of the seedlings as measured by length is shown in Figure 1. For the sake of brevity data are shown only for those seedlings which grew in soil drenched with concentrations of .05, .1, .2, .3, .4, and .5% Systox, and for only three periods of growth 6, 18, and 22 days after seeding. Although no further growth measurements were made after 22 days, the plants continued to grow for a total of 31 days at which time the soil was removed from the roots by washing and examined for nematode infection.

Figure 1 shows differences in growth as measured by height of seedlings grown in root-knot infested soil drenched with different concentrations of Systox. Measurements are in inches above or below that of the control for each of the three growing periods.

Growth differences after six days were not significant between the controls and seedlings grown in soil drenched with .05 and .1% concentration of Systox. Seedlings drenched with .2, .3, .4 and .5% Systox made significantly less growth than the control.

After 14 days, seedlings growing in soil drenched at .05 and .1% Systox were significantly better than the controls. There was no significant difference, however, between the controls and seedlings growing in soil drenched with a .2% concentration of Systox. Those growing in soil drenched with .3, .4 and .5% Systox made significantly less growth than the controls.

After 22 days differences were not significant between the controls and seedlings treated at the .05% concentration; however, seedlings treated at the .1 and .2% concentration were significantly better than the controls. Seedlings growing in soil drenched at .3% concentration showed no significant differences in growth from those of the control. Seedlings treated at the .4 and .5% concentration made significantly less growth than the control.

Figures 2 and 3 show the relative size of the cucumber seedlings after 14 days' growth in soil drenched at the various concentrations. Note in Figure 3 the apparent stunting of those seedlings treated at the .2% concentration and above.

A very similar pattern is shown in Table 2 for leaf area index and top weight. Plants growing in soil treated at the .05 and .1% concentration showed significantly better leaf area and top weight than that of the control. There were no significant differences in root weights for the treated and untreated except for seedlings growing in soil treated at the .5% concentration, which made significantly less growth.

After the 31-day growing period an examination of the roots of the cucumber seedlings from the different treatments with Systox revealed that the check plants were severely galled. Seedlings which had grown in soil drenched with a .05% concentration were lightly infected with root knot, while those in soil drenched with a .1% concentration and above showed no sign of root-knot infection when examined under the dissecting microscope (Figures 4 and 5).

B. To determine possible toxic residues of Systox in the edible fruit, a separate experiment was carried out. Cucumbers were harvested from seedlings which had grown in sterile soil drenched with 300 cc. of Systox at concentrations ranging from .1 to .2%. Only those cucumbers from the .19% treatment were analyzed[2], and these were found to contain .3 ppm of an anticholinesterase inhibitor.

[2]Analysis was made by Dr. John P. Frawley of the Food and Drug Administration. The sample was analyzed for active anticholinesterase residue by a biological procedure, based on extraction, oral administration to flies, and measurement of the inhibition of fly cholinesterase.

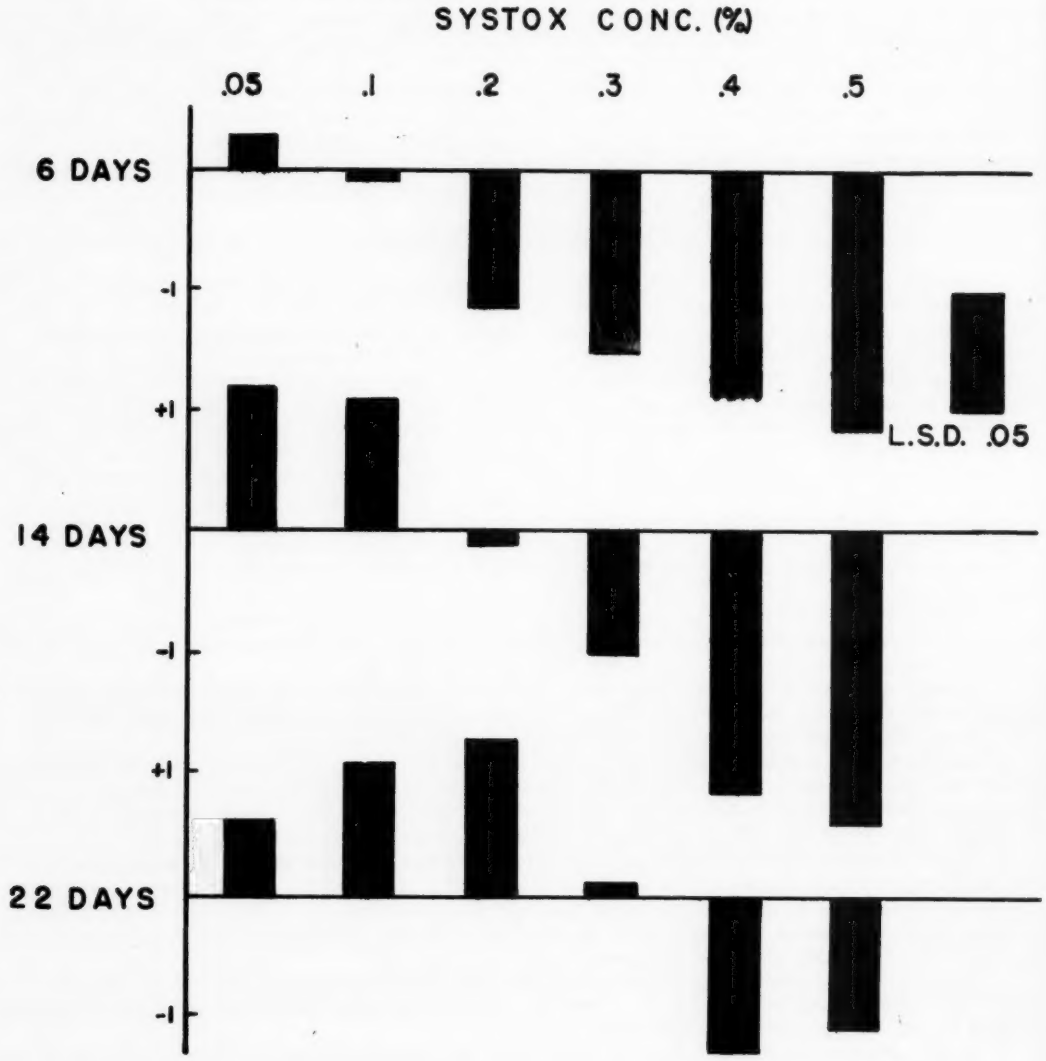

FIGURE 1. Height of cucumber seedlings grown in root-knot infested soil drenched with different concentrations of Systox. Differences are shown in inches above or below height of the controls.

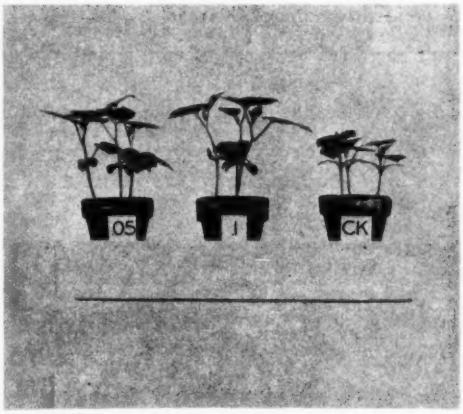

FIGURE 2. Relative size of cucumber seedlings after 14 days growth in soil treated at the lower concentrations of Systox and in untreated soil.

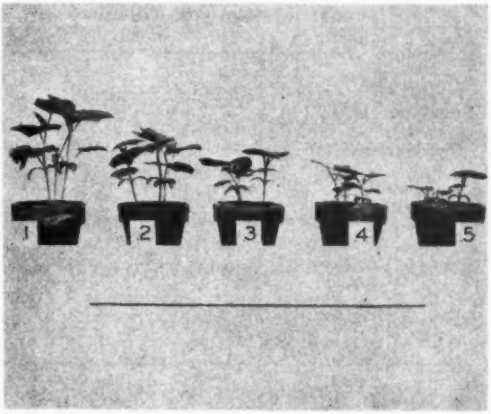

FIGURE 3. Relative amounts of toxicity to cucumber seedlings after 14 days growth in soil drenched at the higher concentrations of Systox.

FIGURE 4. Roots of cucumber seedlings which grew for 31 days in root-knot infested soil not treated with Systox.

FIGURE 5. Roots of cucumber seedlings which grew for 31 days in root-knot infested soil drenched one week prior to seeding with a .1% concentration of Systox. Note clean uninfected roots.

Table 2. Leaf area, top weight, and root weight of cucumber seedlings grown in root-knot infested soil drenched with different concentrations of Systox.

Systox concentration (%)	Leaf area index[a] -- mean of 5 plants			Top weight (gms)	Root weight (gms)
	14 days	18 days	22 days		
Control	41.5	57.0	74.7	28.0	24.6
.05	76.9**	84.6**	92.9**	41.2**	30.2
.1	70.2*	76.4**	92.8**	37.7**	28.6
.2	49.5	68.0*	86.9*	33.6**	23.6
.3	28.5	52.7	78.3	29.0	21.9
.4	25.8**	46.0*	77.4	28.2	23.8
.5	25.3**	46.2*	82.3	25.4	15.1*
L. S. D. .05	10.7	- - -	- - -	5.59	8.19
L. S. D. .01	14.2	- - -	- - -	7.54	11.05

* Significance at .05 level
** Significance at .01 level

[a] Length in centimeters of each leaf was multiplied by each breadth to obtain leaf area index

C. Another experiment was set up using non-inoculated soil, to determine if there was any stimulation from Systox alone which would account for the better growth observed in seedlings grown in soil drenched with concentrations of .05 and .1%. Five-inch pots of sterile soil were drenched with 300 cc. of Systox at these concentrations. Untreated controls were included. Cucumber seedlings planted in the treated pots did not show any significant growth differences from those of the control.

D. Two additional experiments were set up to determine the manner in which the nematodes were being controlled, that is, whether the chemical was acting as a contact poison, intestinal poison, nervous poison, or whether the nematodes were killed at all.

Tomato seedlings were allowed to grow for four days in sterile soil which had been drenched at the time of planting with 300 cc. of Systox in concentrations of .05, .1, and .2%. All treatments were replicated five times and controls were included in which the soil was not drenched. After the four-day exposure period the seedlings were removed and the roots washed free of all soil. The seedlings were then transplanted into soil infested with root-knot nematodes and allowed to grow for 35 days. An examination of the root systems at that time revealed that there was no detectable difference in the amount of root-knot infection between the treated and untreated tomato seedlings. All were moderately infected.

In a second experiment, six egg masses of the root-knot nematode were placed in each of three concentrations of Systox: .05, .1 and .2%. Well-slides were used for convenience in manipulation. A control was included in which tap water was used instead of the Systox solution. The slides were kept in a moist chamber in the laboratory. After a period of one week, one egg mass from each treatment was transferred to another slide containing only tap water. The remaining five egg masses were placed in pots of sterile soil with tomato seedlings which were allowed to grow in the greenhouse for 39 days.

Daily examinations of the slides revealed that no hatching occurred in any of the treatments except the control, in which hatching occurred within 24 hours with most of the eggs hatching by the end of one week. Only two motionless larvae were seen in the case of the other treatments but these probably were already hatched before the chemical was added. Egg masses taken out of the Systox solutions and placed in tap water hatched readily within 24 hours and continued to hatch for several days. Egg masses taken from the Systox solutions and placed in pots with tomato seedlings caused severe infection in 39 days.

Discussion and Conclusions

Systox used as a soil drench in concentrations ranging from .005 to 1.0% caused some reduction of infection and reproduction of root-knot nematodes on tomato seedlings. From the standpoint of root-knot control with little or no phytotoxicity the .1% concentration proved most favorable. Egg masses produced on tomato plants which had grown in treated soil, when used to inoculate cucumber seedlings, were highly infective and apparently were not injured by the chemical.

Cucumber seedlings which grew in soil drenched at the .05 and .1% concentrations made significantly better growth than the controls as measured by height, leaf area, and top weight. In the experiments with cucumbers, root knot was apparently controlled at the .1% and higher concentrations, but the chemical was phytotoxic when applied at concentrations greater than .1%. The better growth rate of seedlings treated at the .05 and .1% concentrations is considered to be due to the control or reduction in activity of the nematodes, since there was no measurable stimulation in growth of cucumber seedlings grown in treated sterile soil.

The minimum rate of application as a drench at which the root-knot nematodes were apparently controlled was 300 cc of a .1% solution per 5-inch pot. This would be equivalent to .3 cc of active ingredient per pot or 8.1 cc active ingredient per cubic foot of soil.

The exact manner in which the chemical controlled the nematodes was not determined. It was shown, however, that the nematodes were not killed by feeding on tomato seedlings which had absorbed Systox from sterile soil. It was also shown that, even though larval hatching from egg masses placed in .05, .1, and .2% Systox solutions was inhibited for one week, hatching occurred readily when the egg masses were removed and placed in tap water. It appears then that control was due both to inhibition of hatching of larvae and also, probably, to paralysis of hatched larvae in the soil. There was some evidence that larvae could be killed by several days' exposure to these concentrations of the chemical, but it is quite certain that eggs were not seriously injured by being immersed for one week in even the .2% solution of Systox.

The inhibition of hatching of larvae from eggs and the apparent paralysis of hatched larvae exposed to Systox may have been due to the destruction of cholinesterase in the nematode, although the presence or absence of cholinesterase in Meloidogyne has not been investigated. Its presence, however, has been demonstrated in the nematode Ascaris megalocephala Cloq. (1) but it was found not to occur in Ascaris lumbricoides Linn. (2).

An organic analysis of cucumber fruit taken from plants which had grown in soil treated at 19% concentration, revealed the presence of .3 ppm of an anticholinesterase inhibitor. This would strongly suggest the presence of Systox in the cucumbers, as there are no degradation products of this compound known which would inhibit cholinesterase. The presence of a cholinesterase-inhibiting compound in the cucumbers would indicate a definite potential toxicity to human beings, since the general pattern of toxicity would be the same in man as in insects.

In view of these findings it must be emphasized that Systox cannot be recommended for control of nematodes until more definite information is available relative to its translocation in plants and possible toxic residues.

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DIVISION OF NEMATOLOGY

TEMPORARY STERILIZATION OF SOIL IN THE EVALUATION OF ROOT AND CROWN ROT LOSSES WITH WHEAT AND BARLEY UNDER FIELD CONDITIONS

G. W. Bruehl

Partial sterilization of field soil with chloropicrin was undertaken in an attempt to determine the effects of soil-borne root and crown rots in aggregate upon the yield of wheat and barley. Others (1, 2, 4) reported large increases in yield following such treatments.

Materials and Methods

The experiments were conducted on the Experiment Station Farm at Brookings, S. Dak., on fertile, dark, well-drained loam with a history of general farming. Chloropicrin was injected at the rate of 3 ml. per square foot at a depth of six inches in recently plowed soil. A strip of land 100 by 13 feet was treated each spring as soon as the soil was warm, and immediately covered with sisalkraft paper to retain the vapors. The paper was removed one week later to permit aeration for about one week prior to planting. An adjacent untreated plot served as a check. Common wheat, durum wheat, and barley were sown across the treated and check soil in rows one foot apart. Four replications of three-row plots were the minimum planting in any year. Contamination of treated soil occurred throughout the season from cultivations, wind, rain, etc. An exploratory number of isolations were made to determine the kind and abundance of invading organisms. Dry weights of plants about 47 and 61 days after planting were taken in 1950 and 1951, respectively, and grain yields were recorded from the center row of each plot each season.

Results

Emergence was delayed and early seedling development retarded as a result of the chloropicrin treatment. In later stages the plants on treated soil developed rapidly until they overtook and exceeded the growth of the plants on the check soil. Plants in the jointing stage on treated soil were taller, had wider leaves, and produced two to three times the dry weight of plants on check soil (Table 1), and they also headed earlier. After about the heading stage final development weakened noticeably. This comparative debility on treated soil was so marked in 1949 that yields were lower than on untreated soil (Table 1). The straw of mature grain on treated soil lacked the bright yellow sheen of healthy straw. The kernel weight of durum wheat in 1949 was reduced from 32.7 grams per 1000 to 24.5 grams on treated soil. Whiteheads did not occur, but many tillers produced no grain. Crowns on treated soil were severely rotted, while those on check soil were comparatively healthy.

Isolation tests showed a build-up of Fusaria in the treated soil from a low level at early stages of growth to abundance. Helminthosporium sativum was more abundant in check crowns than in crowns from treated soil. Trichoderma sp. was more abundant in treated than check host tissues. Gloeosporium bolleyi and bacteria were more abundant in root tissues in treated than check soil. Sample isolation data are presented in Table 2, a and b.

Discussion

When chloropicrin was applied to field soil wheat and barley responded by an initial retardation, then a period of accelerated growth, and a comparative deterioration in the head filling stages. Toxic conditions following treatment accounted for the initial retardation. The subsequent rapid growth probably was due to two main causes: (1) an increase in available nutrients, and (2) a relative absence of soil-borne pathogens. The final deterioration following heading may be attributed to a multiplication of soil-borne pathogens resulting in severe crown rot. In treated soil the Fusaria increased from a very low level to abundance in a few months. Helminthosporium sativum was more abundant in crowns from check plants, indicating its slow invasion of the treated soil.

The etiology of the late stage deterioration is merely speculative until a more complete investigation can be made, but Fusaria and Trichoderma sp. constituted a larger proportion of the isolates on treated than on check soil. It would be logical to consider that their increase brought on the late stage deterioration. Edwards (3) found that Trichoderma viride (Pers. ex Fries) Bisby

Table 1. Dry weights of plant tops harvested in two intermediate stages of growth, and grain yields at maturity of both wheat and barley grown on treated and untreated field plots.

Season	Planting date	Crop variety	Dry weight of shoots on treated soil in percent of check (Days after seeding: 47)	Dry weight of shoots on treated soil in percent of check (Days after seeding: 61)	Yields, bushel per acre: Check	Yields, bushel per acre: Treated	Yields, bushel per acre: Percent increase or decrease	Harvest date
Common wheat -- average gain of grain 23 percent								
1948	5/3	Mida	---	---	15.6	18.5	+19	8/5
1949	5/3	Rushmore	---	---	17.9	17.2	- 4	7/28
1950	5/1	Rushmore	193	172	12.8	19.0	+48	8/9
1951	5/26	Lee	301	311	18.6	24.2	+30	9/4
Durum wheat -- average loss of grain 3 percent								
1948	5/3	Mindum	---	---	20.7	18.3	-12	8/15
1949	5/3	Mindum	---	---	18.0	9.7	-46	8/9
1950	5/1	Mindum & Peliss	192	188	16.3	19.7	+22	8/21
1951	5/26	Ld. 306	234	361	13.5	17.0	+26	9/4
Barley -- average gain of grain 21 percent								
1948	5/3	Wisconsin Barbless	---	---	26.5	26.1	- 2	7/28
1949	5/3	Odessa	---	---	50.9	43.4	-15	7/23
1950	5/1	Odessa & Feebar	236	174	32.1	42.1	+40	8/5
1951	5/26	Velvon 11	250	228	33.3	53.6	+61	8/25

became a virulent parasite on maize seedlings in the absence of inhibitors. Whether the Fusaria largely accounted for the crown rot, or whether Trichoderma sp. assumed the role of a major parasite subsequent to the soil treatment is not known.

Whatever the nature of the crown rot, it was more severe in warm seasons (Table 3).

The severity of the late stage deterioration was greatest on durum wheat with losses of grain of 12 percent and 42 percent, respectively, in 1948 and 1949. Common wheat suffered slightly in 1949, whereas barley yields were reduced 15 percent by treatment in 1949.

We are approaching the time when the chemical industry may provide economic and practical soil fungicides for use on farm lands. Large increases in yields of barley and wheat are possible as indicated by dry weights at intermediate stages of growth (Table 1). The failure to improve grain yields in some seasons indicates a need of caution. However, these experiments may have permitted excessive reinvasion because the treated strip was only 13 feet wide. Destruction of the normal balance of microflora can be devastating under conditions favorable to contamination as indicated by a 46 percent loss in yield of durum wheat in 1949.

The data presented did not provide an estimate of root and crown rot losses in wheat and barley.

Table 2a. Fungi isolated[a] on water-agar from small grain roots six weeks after planting, 1950.

Host	Soil	Isolates[b] H	F	G	T	C	R	M	P	N.B.
Common wheat	Treated	0	2	7	5	5	0	5	1	28
	Check	0	17	6	0	7	0	6	4	11
Durum wheat	Treated	0	0	7	0	1	0	0	0	19
	Check	1	11	3	1	1	0	0	0	9
Barley	Treated	0	0	9	1	0	0	0	0	16
	Check	0	11	1	0	1	1	0	0	17
Totals	Treated	0	2	23	6	6	0	5	1	63
	Check	1	39	10	1	9	1	6	4	37

Table 2b. Fungi isolated on acidified-potato-dextrose agar from crown tissues one week prior to harvest, 1951[c].

Host	Soil	Isolates[b] H	F	G	T	R	M	Ph	A	Pe	X
Common wheat	Treated	11	14	8	7	0	0	5	3	0	29
	Check	23	15	5	1	1	0	4	4	3	39
Durum wheat	Treated	7	33	5	9	0	3	5	3	0	49
	Check	25	11	4	0	0	0	4	1	0	37
Barley	Treated	10	18	5	6	2	0	1	0	0	36
	Check	30	14	4	0	0	1	0	0	0	37
Totals	Treated	28	65	18	22	2	3	11	6	0	114
	Check	78	40	13	1	1	1	8	5	3	113

[a] Roots washed in sterile water, blotted, plated.

[b] H = Helminthosporium sativum Pam., King, & Bakke; F = Fusarium spp.; G = Gloeosporium bolleyi Sprague; T = Trichoderma sp.; C = Cephalosporium sp.; R = Rhizoctonia solani Kuehn; M = Mucoraceae; P= Pythium spp.; N.B. = negative or dominated by bacteria; Ph = Phoma sp.; A = Alternaria sp.; Pe = Penicillium sp.; X = number of crown pieces plated.

[c] Crowns were stripped, base of primary axis removed, wet in 70% ethyl alcohol, soaked in 5.25% sodium hypochlorite for 30 seconds, and plated.

Table 3. The effect of rainfall and temperature on disease development in cereal plots treated with chloropicrin, 1948-1951, Brookings, South Dakota.

	1948	1949	1950	1951
Mean monthly rainfall (inches)[a]	2.89	1.72	2.43	4.07
Mean monthly temperature (^{o}F)[a]	63.4	65.4	58.1	59.3
Yield differences in the three test cereals in response to chloropicrin treatment (percent)	+2	-21	+37	+32

[a] From Monthly Climatological Data, Weather Bureau, U. S. Department of Commerce.

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DIVISION OF CEREAL CROPS AND DISEASES, U. S. DEPARTMENT OF AGRICULTURE AND DEPARTMENT OF PLANT PATHOLOGY, SOUTH DAKOTA STATE COLLEGE, BROOKINGS, SOUTH DAKOTA

STEWART'S DISEASE PROSPECTS IN ILLINOIS FOR 1952

G. H. Boewe

In 1952, Stewart's disease or bacterial wilt (Bacterium stewarti) of corn probably will occur much farther north and be much more destructive in Illinois than it was in the summer of 1951.

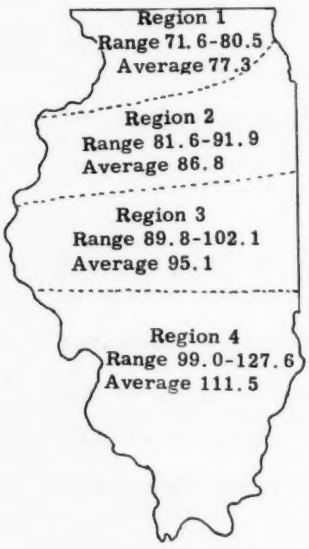

FIGURE 1. Winter temperature indices (see text) for Illinois, 1951-52.

Two principal types of damage to corn are produced by Stewart's disease: an early-season wilt and death of plants, especially in sweet corn, and late-season leaf blight, which may be severe enough to cause death of plants, on both sweet corn and field corn. The wilt and leaf blight phases of the disease are expected to appear on susceptible varieties of corn in Illinois in 1952 as shown by the table below.

Region	Early-season wilt	Late-season leaf blight
1. Extreme north	None	None or trace
2. North	Absent or nearly so	Light to moderate
3. Central	Light to moderate	Moderate to severe
4. South	Destructive	Severe

The four regions listed in the table above are delineated on the map (Figure 1). These regions have been determined on the basis of winter temperature indexes (the sum of the mean temperatures of December, January, and February) calculated from the records of 89 weather stations distributed throughout the State. The data were obtained from the U. S. Weather Bureau office at Springfield, Illinois. The range of indexes and an average index are shown for each region. As indicating the future occurrence of Stewart's disease, these indexes appear to have significance, as follows:

If the index is	Early-season wilt probably will be	Late-season leaf blight probably will be
100 or more	Destructive	Severe
Between 90 and 100	Light to severe	Severe
Between 85 and 90	Absent or nearly so	Moderate to severe
Between 80 and 85	Absent or nearly so	Light to moderate
Below 80	Absent or nearly so	A trace, at the most

This forecast on Stewart's disease, the fourth to be made for Illinois, is based on the close relationship that appears to exist between the amount of disease which develops during the summer and the temperature of the preceding winter. The development of both phases of the disease on sweet corn during the past three years coincided very closely with the predictions made in the first three forecasts. The development of the leaf-blight phase on field corn has also coincided very closely with our previous predictions.

ILLINOIS STATE NATURAL HISTORY SURVEY, URBANA

BLACK RASPBERRY MOSAIC IN WESTERN WASHINGTON

Glenn A. Huber and Folke Johnson[1]

In former years, culture of black raspberries was a thriving industry in western Washington comprising approximately 1,000 acres. In the middle thirties there was a sharp decline in production and commercial black raspberry growing was practically abandoned. Surveys by the senior author revealed that a virus disease was the cause of this sharp decline in production. The disease was widely distributed in most of the areas where black raspberries were grown. It was observed that in both commercial and home plantings, wherever black raspberries were planted adjacent to red varieties virus symptoms were pronounced on the black raspberries. On the other hand, no such expressions were present in the red varieties except for an indistinct chlorotic "flecking" or "blotching" on the lower leaves of current season's canes. These mild symptoms on the red raspberries had generally been considered to be caused by low temperatures, nutrient deficiencies, or other physiological disturbances.

Because of this condition, an investigation was conducted in which it was found that mosaic in black raspberries was caused by a different virus or complex of viruses than that causing the common green mosaic in the red varieties. Experiments revealed that the chronic symptoms of "flecking", herein referred to as mild mosaic of red raspberries, were in reality expressions of the black raspberry mosaic virus.

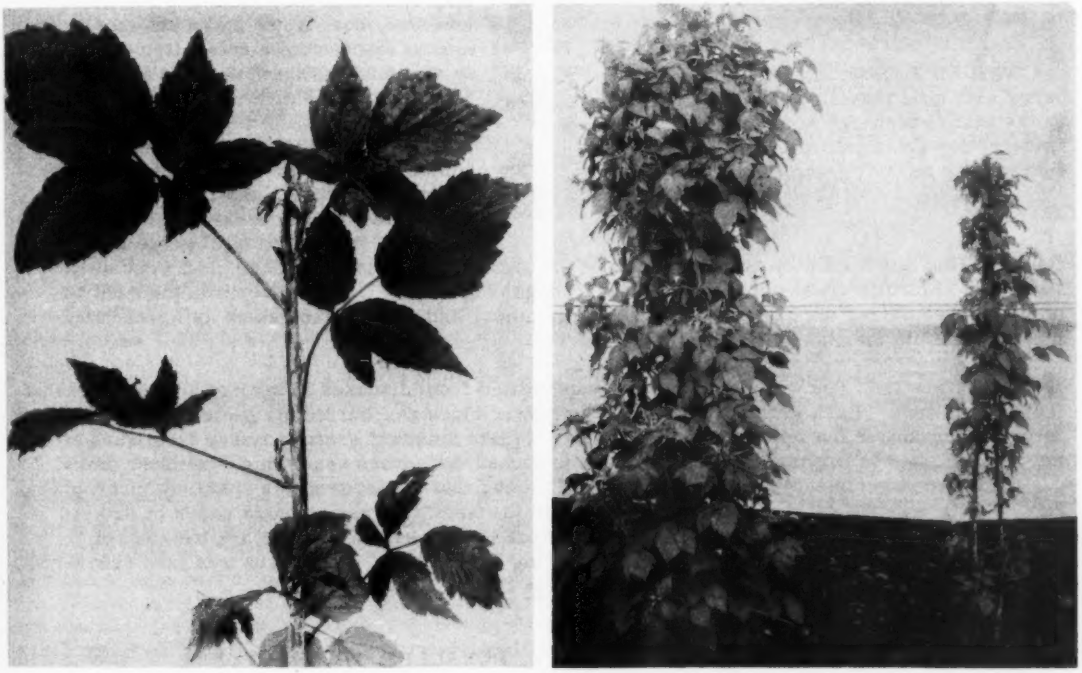

FIGURE 1. Left. Shoot of Cumberland black raspberry showing symptoms of tip-dieback, necrosis along the stem and mosaic in the terminal leaves. Right. Healthy and infected Cumberland plants, respectively, showing the dwarfing effect of the virus.

Symptoms: In black raspberry varieties, the disease is manifested by two types of symptoms: cane-tip dieback and leaf mottling. Primary symptoms appear on infected plants as small,

[1]Formerly and present plant pathologist at the Western Washington Experiment Station, Puyallup, Washington.

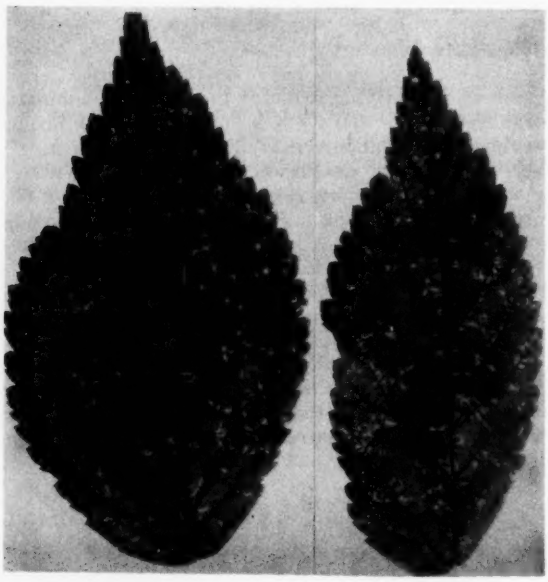

FIGURE 2. Two leaflets of Cuthbert red raspberry with mild mosaic symptoms of flecking caused by the black raspberry mosaic virus. There is a tendency for these chlorotic flecks to form rings surrounding normal green tissue.

water-soaked areas or streaks at the tips of current season's canes or on the young leaf petioles. These water-soaked areas become purple in color and the cane tips bend downward, become brittle, and snap off easily when pressure is applied. It is not unusual for the terminal portions of these canes to die. In some instances during the high summer temperatures, growth is checked without cane-tip distortion. Subsequently, growth again commences leaving a rosette of leaves at that point. Leaf mottling becomes evident on young leaves by the appearance of light green areas intermingled with the normal green tissue. These chlorotic areas occur irrespective of leaf veins and may assume an irregular ring-spot pattern. The mosaic symptoms are transient, disappearing during hot weather and reappearing when temperatures moderate later in the growing season. Either cane-tip dieback or leaf mottling may appear in the absence of the other; however, both types of symptoms eventually appear on affected plants. Growth is greatly retarded and the fruiting laterals fail to attain the length of those on healthy plants (Figure 1).

In red raspberries the symptoms are somewhat indistinct. During the cool temperatures of spring and fall, the disease is manifested by the appearance on the leaves of small, irregular, light green areas or flecks. This slight mottled condition is most pronounced on the lower foliage, and in some varieties these chlorotic areas appear in indistinct rings (Figure 2). The symptoms in the field become masked during the high temperatures of summer, and the disease has no apparent detrimental effect on the plants.

Experimental: The virus has not been transmitted from diseased to healthy plants by mechanical inoculations. Bark grafts from severely affected black raspberries to healthy red varieties invariably produced the mild mosaic symptoms. In like manner, grafting tissue from such affected red varieties to healthy black raspberries produced the severe symptoms described above.

Repeated experiments by the senior writer showed that the large green aphid, Amphorophora rubi, Kalt., was the natural vector of the virus in the field. By using aphids under controlled conditions, the virus was transmitted from the black to the red raspberries and back again, with the characteristic disease symptoms appearing in the plants. The virus was also recovered from the wild black raspberry, Rubus leucodermis Dougl.

WESTERN WASHINGTON EXPERIMENT STATION, PUYALLUP, WASHINGTON

OCCURRENCE OF YELLOW-NET VIRUS DISEASE IN TABLE BEET SEED PLANTS IN NORTHWESTERN WASHINGTON[1]

D. M. McLean

Sylvester (4), in 1948, reported the occurrence of yellow-net, a virus disease of sugar beets (Beta vulgaris L.) in northern California. He described the symptoms and showed that the virus has a limited host range, infecting all varieties of sugar beets and garden beets (B. vulgaris) tested, as well as Swiss chard (B. vulgaris var. cicla L.). Among the several species tested in a number of families, spinach (Spinacia oleracea L.) and Turkish tobacco (Nicotiana tabacum L.) failed to develop symptoms. The virus can be transmitted by several aphid species (5) but is not mechanically transmitted by ordinary methods (4). Diseased specimens found in the field were limited in number and relatively isolated. The disease apparently has a limited distribution in commercial sugar beet fields in southern California, and it has been observed in commercial sugar beet plantings in England[2].

Yellow-net appeared in a number of table beet seed plants in an experimental planting at the Northwestern Washington Experiment Station, Mount Vernon, in 1951. The stecklings had been grown in 1950 in several locations well isolated from the general seed-producing area, as recommended for control of mosaic in seed crops (1). This practice has made possible practical control of mosaic by the elimination of viruliferous aphids from infected seed plants in the root beds. The yellow-net infected plants found were not all from the same root bed location. Careful inspections of a number of steckling beds were made during the 1951 season, and infected plants were found in two locations.

Unusual virus-like foliar symptoms are conspicuous in table beet seed crops. Because early symptoms of yellow-net might readily be confused with those of mosaic, diseased specimens were taken into the greenhouse for observation and transmission studies. Similar specimens were sent to G. H. Coons at Beltsville, Maryland, and to N. J. Giddings at Riverside, California, where typical symptoms of yellow-net developed.

The virus was not transmissible with juice extract from diseased plants by use of carborundum, a method which is very effective in the transmission of beet mosaic virus (1, 2). The virus is readily transmitted to garden beets, sugar beets, Swiss chard, and mangel (B. vulgaris) by using 5 to 10 infectious green peach aphids [Myzus persicae (Sulz.)]. Typical severe vein yellowing developed in all the suscepts and differed only in severity.

Specimens of sugar beets containing viruses of both mosaic and yellow-net were sent to the writer from Berkeley, California, by E. S. Sylvester. These specimens made possible a comparison of mosaic and yellow-net from two sources (California and Washington) and a study of the virus-complex symptoms. The mosaic virus was readily isolated from the complex by manual transmission with extracted juice to garden beets, sugar beets, Swiss chard, mangel, zinnia (Zinnia elegans Jacq.), and spinach (Hort. var. Bloomsdale). There was no discernible difference in the symptoms caused by mosaic virus from the two sources, as concluded by Pound (1).

It was shown by Severin and Drake (2), Sylvester (6), and Watson (8) that the mosaic virus is non-persistent in aphid vectors. Aphid vectors are not infectious three hours after infection feeding (2). Vector efficiency is enhanced by pre-infection fasting followed by short multiple infection feedings (3, 6, 8). Yellow-net virus, according to Sylvester (4, 7), is persistent within the aphid vectors. Following infection feeding, the green peach aphid remained infectious for 11 days, the period of its life under the experimental conditions (4).

In transmission studies at Mount Vernon, the yellow-net virus was readily separated from the mosaic virus in the California sugar beet virus complex either by a post-infection fasting of three hours or longer, or by transferring viruliferous aphids from sugar beet to any of the suscepts and after three hours again making a serial transfer of the aphids to another set of suscepts. By using spinach or zinnia in the first transfer, it was possible to transfer mosaic virus only (since these species are not susceptible to yellow-net) and on the second transfer (after three hours) to lose the mosaic virus and transfer only the yellow-net virus. Aphids are much more efficient vectors of yellow-net virus than of mosaic virus, as shown by Sylvester (5). Aphids frequently fail to transmit the mosaic virus when transferred by a brush from an infected plant to a healthy suscept.

[1]Cooperative investigations of the Division of Vegetable Crops and Diseases and the Northwestern Washington Experiment Station, Mount Vernon, Washington.

[2]Correspondence with C. W. Bennett, Division of Sugar Plant Investigations, Riverside, California, and with R. Hull, Rothamsted Experiment Station, Dunholme Holt, Lincoln, England.

From a comparison of symptoms and suscept range, it was concluded that the yellow-net observed in northwestern Washington is similar to the yellow-net reported from California and probably is caused by the same virus. The following species failed to show symptoms after inoculation with aphids: Zinnia elegans, Fragaria vesca L., Gladiolus sp., Dahlia pinnata Cav., chickweed [Stellaria media (L.) Cyrill.], and crownbeard [Verbesina encelioides (Cav.) B. & H.]. Only plants in the genus Beta have been experimentally infected with yellow-net virus.

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DIVISION OF VEGETABLE CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS AND AGRICULTURAL ENGINEERING, AGRICULTURAL RESEARCH ADMINISTRATION, U. S. DEPARTMENT OF AGRICULTURE, AND NORTHWESTERN WASHINGTON EXPERIMENTAL STATION, MOUNT VERNON, WASHINGTON

EXCEPTIONAL HEAT TOLERANCE AND SOME OTHER PROPERTIES OF THE CHRYSANTHEMUM STUNT VIRUS

Philip Brierley

Since the chrysanthemum stunt virus was found to be manually transmissible[1], efforts have been made to determine its thermal inactivation point. Seven experiments have failed to define such an end point. In two tests infected leaves were ground in a mortar with 1 to 4 times their weight of distilled water; in five tests similar preparations were macerated in a blendor. The resulting suspensions were strained through two layers of cheesecloth without pressure before they were heated in blood tubes enclosed in a water bath at various temperatures for 10 minutes. In four experiments Mary MacArthur was the source and test variety; in the other tests Mistletoe chrysanthemums served as source and test plants. In the tests with the Mary MacArthur variety an inactivation point at about 98°C was suggested, but in later trials with Mistletoe no such end point was indicated. Results from other experiments suggest that Mistletoe is superior to Mary MacArthur as a source of the stunt virus, or as a test plant, or as both.

When a water-bath temperature of 100° C failed to inactivate the virus, similar suspensions were boiled for 10 minutes. In the first boiling trial, 100 gm. of young leaves were macerated in a blendor with 250 ml. of distilled water and the product was strained as before. A 50-ml. sample was heated for 10 minutes in an open flask over a gas flame. As the crude extract foamed strongly, it was not feasible to boil this sample continuously. After cooling, the heated sample was withdrawn with a pipette to a clean flask and used to inoculate Mistletoe chrysanthemums. Eight of the 10 plants inoculated developed typical stunt symptoms. In a second trial, 75 gm. of infected Mistletoe leaves were macerated with 225 ml. of distilled water, and a 100-ml. sample was then boiled for 10 minutes. Addition of 1 ml. of paraffin oil reduced foaming but did not eliminate it. Eighteen of 20 Mistletoe plants inoculated with this boiled sample developed stunt. In a third trial, 25 gm. of affected Mistletoe leaves were ground in a mortar with 100 ml. of distilled water. After straining, a 50-ml. sample was boiled for 10 minutes as before. Addition of 2 ml. of 95% alcohol and 1 ml. of paraffin oil eliminated foaming and permitted active boiling for the full period, resulting in loss of over half of the original volume of the preparation. After cooling, 15 ml. were pipetted into a clean flask and used a inoculum. One of 25 Mistletoe chrysanthemums inoculated developed typical stunt symptoms.

Not only does the chrysanthemum stunt virus withstand boiling, but some boiled samples prove as infectious as unheated samples of the same virus preparation. Such heat tolerance is exceptional, for other relatively stable plant viruses are inactivated by 10 minutes heating at 90° to 95° C. A virus of sunflower (Helianthus annuus L.) in Argentina[2] has been reported to withstand 98° to 99° C. Although sunflower is a near relative of chrysanthemum we have been unable to infect it with the chrysanthemum stunt virus.

When leaves of stunt-affected Mary MacArthur were allowed to dry in an open tray in a greenhouse the virus remained infective for only two to four weeks in three trials. In two similar trials with leaves of Mistletoe chrysanthemums, however, the virus was still infective after eight weeks, the longest period tested. In two trials of dilution, preparations similar to those used in heat-tolerance tests were infectious after diluting 1 to 10,000; much less dilution was tolerated in four other trials.

Comparatively low tolerance of dilution indicates that the extracts were low in virus concentration. A low concentration of the virus in chrysanthemum leaves seems unlikely in view of the rapid dissemination of the virus during commercial handling of the plants. Possibly water is a poor extractant and diluent for this virus. Leaf extracts stored in open tubes at 18° C proved infectious after 7 weeks' but not after 8 weeks' aging in four trials. Inactivation of the virus was probably accelerated by molds which entered the open tubes, for other viruses in crude sap withstand such aging longer than they withstand drying.

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[1]Brierley, Philip, and Floyd F. Smith. 1949. Evidence points to virus as cause of Chrysanthemum stunt. Florists' Review 103 (2676): 36, 37.

[2]Traversi, Blanca A. 1949. Estudio inicial sobre una enfermedad del girasol (Helianthus annuus L.) en Argentina. Rev. Invest. Agri. B. Aires 3, 4: 345-351.

Because dipping tools in alcohol and flaming them constitute a common precaution against dissemination of the stunt virus, one experiment on resistance to alcohol was made. Stunt-affected Mistletoe leaves were macerated in a blendor with three times their weight of distilled water, or of 95% alcohol. After standing for one hour the alcohol preparation was strained, diluted with water to 20% alcohol content and used as inoculum. Nine of 10 Mistletoe plants developed stunt after inoculation with the alcohol preparation and a like number after inoculation with the water preparation. Reliance should be placed in the flaming rather than in the alcohol dip for sterilizing tools against the stunt virus.

U. S. BUREAU OF PLANT INDUSTRY, SOILS AND AGRICULTURAL ENGINEERING, BELTSVILLE, MARYLAND

DISEASES OF PEPPERMINT IN OREGON IN 1951

C. E. Horner

Peppermint (Mentha piperita var. Mitcham) is grown on approximately 15,000 acres in Western Oregon. Surveys of the major mint growing areas have revealed four fungus diseases of this crop. Two of these diseases have not previously been reported on peppermint in Oregon.

Peppermint Rust: An epiphytotic of peppermint rust (Puccinia menthae Pers.) during 1949 and 1950 has been reported (1). Mint rust was present in all the peppermint growing areas in Oregon in 1951. Losses in Columbia County were 25 to 35 percent in experimental plots, and estimated losses for the County were about the same. The mint growing areas in Columbia County are adjacent to the Columbia River about 40 miles from the Pacific Ocean. The area is one of considerable summer fog, high humidity and strong winds. These conditions favor the build-up and spread of mint rust.

Rust was not so severe on peppermint in Lane, Linn, Marion, and Yamhill Counties in the Willamette Valley, where the estimated loss was about 5 percent for the area. The summer of 1951 in the Willamette Valley was not favorable for mint rust development. Precipitation during June, July and August was 0.21 inches while the 60-year average for the same months is 1.85 inches (2). Hot, dry weather prevailed throughout the growing season.

Verticillium Wilt: Verticillium wilt (Verticillium albo-atrum Reinke & Berth.) was first reported on peppermint in Oregon in 1944 by Boyle (3). A thorough survey of the mint growing areas in 1949, during which suspicious appearing plants from 33 farms were examined, resulted in no isolations of Verticillium. In 1951, V. albo-atrum was isolated from peppermint in five fields in Marion County. The disease was not general in any of the fields where it was observed. Plants showing symptoms were in all cases confined to small spots.

Root and Rhizome Rot: Steenland and Burke (4) reported a species of Typhula causing a root rot of mint in Polk County, Oregon in 1950. Surveys of all mint producing areas during January, February, and March, 1951, revealed that rhizomes, young shoots and roots were being damaged by a progressive necrosis. In many cases the necrotic areas were progressing up the new shoots from the parent rhizome. In other cases the new shoots were attacked two to six inches below the ground, causing a type of damping off.

Isolations revealed a complex of several fungi to be associated with this condition. Rhizoctonia solani, Fusarium roseum, Fusarium sp., and Pythium sp. were consistently isolated from advancing necrotic margins. Wound inoculations of healthy rhizomes with each of the above fungi consistently resulted in typical symptoms. Reisolations resulted in the recovery of the fungi from the infected rhizomes. As far as the author is aware, these fungi have not previously been reported on peppermint.

Powdery Mildew: Powdery mildew (Erysiphe sp.) was found on peppermint in three fields in the Willamette Valley but losses were negligible. The fungus has been carried on peppermint in the greenhouse where experimentally it has infected Mentha arvensis var. canadensis (L.) Briq. and Satureja douglasii (Benth.) Briq. Under greenhouse conditions it has been much more severe on the above two plants than on peppermint. No perfect stage of the fungus has been observed. Powdery mildew has not previously been reported on peppermint in Oregon.

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DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, OREGON AGRICULTURAL EXPERIMENT STATION, CORVALLIS, OREGON

UNUSUAL HOSTS FOR TWO SOUTHWESTERN MISTLETOES

Frank G. Hawksworth

Arceuthobium douglasii on Abies concolor

Douglas-fir, Pseudotsuga taxifolia, is usually considered to be the only host of Arceuthobium douglasii, the Douglas-fir dwarf mistletoe.[1] In 1950, the writer found a small white fir, Abies concolor, heavily infected by this parasite in the Sangre de Cristo Mountains near Eagle Nest Lake in northern New Mexico. This tree was about 12 feet high and was surrounded by a heavily infected Douglas-fir overstory; although there were several other white firs in the understory, only one was infected. The parasites were morphologically similar on the two hosts and induced witches' broom formation on both. Arceuthobium campylopodum f. abietinum, the usual dwarf mistletoe on Abies spp., was not seen in this area. White fir is rarely attacked by dwarf mistletoes in the Southwest while Douglas-fir is often subject to severe infection. Arceuthobium douglasii appears to be of no pathological importance on white fir.

Weir[2] recorded A. (Razoumofskya) douglasii on Abies concolor, A. grandis, A. procera, A. lasiocarpa, A. amabilis, and Pseudotsuga taxifolia, but since he regarded Arceuthobium douglasii as synonymous with A. campylopodum f. abietinum (R. douglasii abietina), it is impossible to know for certain with which species he was dealing. Weir was successful in establishing infections of A. douglasii from Douglas-fir on various species of Abies by seed plantings. In addition, it appears that he may have found the true A. douglasii on Abies spp. in nature, at least in some instances, as he inferred that dwarf mistletoe infections on species of this genus were in most cases found in localities where Douglas-fir was heavily infected. As a general rule, the presence of A. campylopodum f. abietinum on true fir is not associated with mistletoe-infected Douglas-fir.

Phoradendron juniperinum on Chamaebatiaria millefolium

Although Phoradendron juniperinum has been reported as confined to the genus Juniperus[3], E. T. Christensen, Assistant Park Naturalist at Grand Canyon National Park, Arizona, found it on tansybush or fernbush, Chamaebatiaria millefolium[4], on the South Rim of the Canyon. As in the previous case, this was an example of a single infected plant growing under an infected stand of a common host, in this instance Utah juniper, Juniperus osteosperma.

DIVISION OF FOREST PATHOLOGY, BUREAU OF PLANT INDUSTRY, SOILS & AGRICULTURAL ENGINEERING, ALBUQUERQUE, NEW MEXICO

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[4] Rosaceae

THE SWEETGUM BLIGHT

Paul R. Miller and G. Flippo Gravatt

Blight, a new disease of sweetgum, Liquidambar styraciflua, was first reported in 1951.[1] The senior author first observed symptoms in the sweetgum trees on his property in University Park, Prince Georges County, Maryland, in 1948. In surveying for the disease in University Park and immediate vicinity in 1949, he found that 20 percent of the 3,000 trees observed showed various stages of decline. The number of trees in different stages of the disease indicated that it had been present for some years. However, numerous residents of University Park stated that they had not observed the disease in former years and there was no evidence that trees had been removed. Local pathologists, foresters, and arborists were consulted but no one had seen this condition in sweetgum trees. In 1949, the senior author also spent several days making an automobile survey in an area of 30 miles radius from University Park but he noted no diseased trees.

Various pathologists have made cultures but have not obtained any organisms that they considered to be the cause of this disease. Further isolation work and inoculations by the Division of Forest Pathology are under way. In local areas, unusual climatic conditions sometimes adversely affect trees, but the progressive symptoms on diseased trees and the increase in the number of trees affected in University Park suggest that the disease may be infectious. The Division of Forest Pathology now has the responsibility for the United States Department of Agriculture for survey and research on this disease.

Symptoms

The first visible indication of blight occurs in late summer; the leaves on affected limbs or small branches show fall coloration much earlier than on healthy branches. Normal, healthy sweetgum trees frequently show some differences in the time of appearance of fall coloration within a tree, but not so strikingly as trees affected by the disease. The following spring, diseased limbs may be dead. On diseased limbs that are not dead leaves that are very small or slightly reduced in size may develop. The small-leaved branches sometimes gradually wilt and then dry up (Fig. 1-B), on others the leaves prematurely become colored red or yellow. Sometimes many of the buds toward the terminal end of such branches remain green and normal in appearance for a while but do not open. Usually the end of the branch or leader, which started growth with small green leaves or failed to produce leaves, gradually dies during the summer and frequently there are brown to black areas in the twig bark.

Affected branches may show up in any part of the periphery of the crown or leader, and rather frequently the whole periphery of limbs will be affected (Figs. 2, 4, 5, 6). On such trees only a few shoots along the main stem will be alive the following season and the entire tree will be dead before fall. Some trees die more rapidly than others, but in general there is a remarkable regularity to the decline and death after the trees show the first symptoms.

Some trees show a general thinness of foliage throughout the tree rather than on only one or two affected limbs. A normal sweetgum has a rather dense foliage, and trees with thin foliage are conspicuous in contrast. On some limbs of these thin, sparse-foliaged trees there are dead tips and gradually dying small leaves. Frequently the leader of the tree is affected first and later symptoms of dying extend on down the tree. This occurs more frequently in sweetgums growing in dense forest stands than on spreading trees growing in the open.

A cut across affected branches and tops reveals amber-brown to deep-brown streaks in the otherwise white satiny sapwood, (Fig. 3). There is no regular pattern of discoloration, sometimes there are just a few streaks and in other cases numerous narrow streaks or broad bands. This discoloration does not normally appear in cross sections of the limbless lower part of the trunks of diseased trees. In diseased sweetgums, some areas in the sapwood appear dryer than others. The heartwood exhibits the normal reddish-brown coloration.

Sweetgum is a very important forest tree in the southeastern quarter of the United States, from Virginia and Tennessee southward and westward to east Texas and east Oklahoma, inclusive. It makes up nearly 8 percent of the total sawtimber (conifer and hardwood) of this region, or 18 percent of the hardwood sawtimber. In 1945, sweetgum sawtimber in this quarter of the country was estimated at nearly 26 billion board feet. As an ornamental and shade tree, sweetgum is outstanding in value in most of the eastern half of the country. Its natural range is shown in Figure 7, but it is planted as an ornamental far north of its natural range and also in southern California.

[1] Miller, Paul R., and Muriel J. O'Brien. An apparently new sweetgum disease in Maryland. Plant Dis. Reptr. 35:295-297. 1951.

FIG. 1

FIG. 3

FIG. 2

FIGURE 1. A -- Healthy sweetgum. B. -- affected by blight. Small, sparse foliage, and curling and browning of the leaves, are some of the symptoms of blight of sweetgum.

FIGURE 2. Blight is slowly killing this large sweetgum.

FIGURE 3. A -- Longitudinal section; B -- oblique section; of sweetgum branch affected by blight, showing irregular discoloration, somewhat more abundantly developed in this case than usual.

FIGURE 4. Blight is rapidly killing valuable sweetgum shade trees in the older infected area of University Park, Maryland.

FIGURE 5. Blight in various stages is apparent in this row of sweetgum trees.

FIGURE 6. Sweetgum trees around this small farm home show various stages of blight.

FIGURE 7. Distribution of sweetgum in the United States. (From U. S. Dept. Agr. Misc. Pub. 287).

Disease Distribution

Except for intensive observations and records indicating spread in University Park for the past three years and illustrated in Figure 8, very little survey work has been conducted for this disease. Numerous blighted sweetgums were found this spring in the vicinity of Aberdeen and Solomons Island, Maryland, and in Caroline and King Counties, Virginia, each nearly 60 miles from University Park, Maryland. F. H. Berry, of the Division of Forest Pathology, recently noted similar symptoms on sweetgum a few miles north of Richmond, Virginia, nearly 100 miles south of University Park. In nearly all of the small stands of sweetgums observed within the affected area, at least a few trees show the first stages of the blight and in some of the stands 5 to 25 percent of the trees show symptoms, usually in an early stage.

Sometimes the diseased trees occur in groups, but they may be randomly distributed among healthy trees. The distribution of affected trees does not appear to be related to soil types. Diseased trees have been observed in open fields where they are growing in fertile, well-drained soil with no grading or other disturbance. The disease has not been observed in trees smaller than about 1 inch in diameter and 6 feet in height.

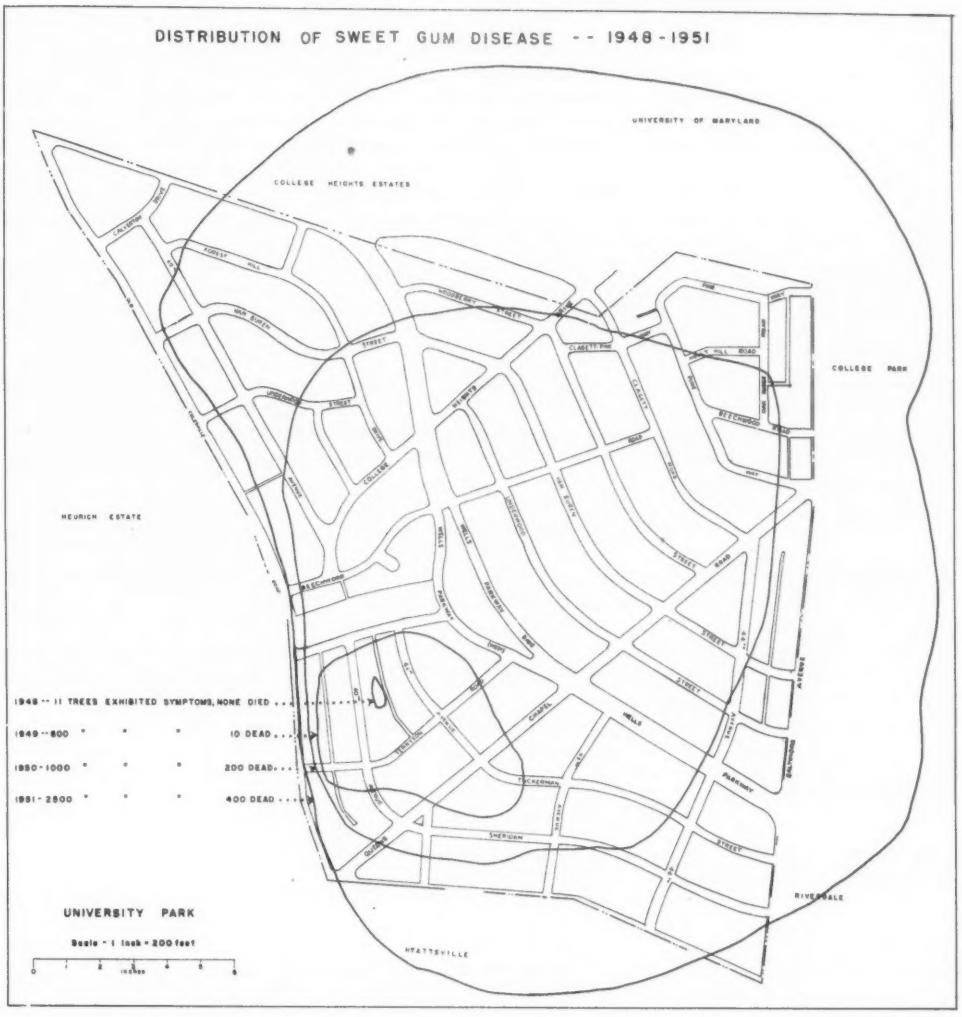

DISTRIBUTION OF SWEET GUM DISEASE -- 1948-1951
UNIVERSITY OF MARYLAND
COLLEGE HEIGHTS ESTATES
COLLEGE PARK
HEURICH ESTATE
1948 -- 11 TREES EXHIBITED SYMPTOMS, NONE DIED
1949 -- 600 " " " 10 DEAD
1950 - 1000 " " " 200 DEAD
1951 - 2800 " " " 400 DEAD
UNIVERSITY PARK
Scale - 1 Inch = 200 feet
RIVERDALE
HYATTSVILLE

FIGURE 8. Distribution of sweetgum blight in University Park, Maryland, 1948-1951.

1948--Symptoms first noted in August on eleven trees within a block. Surveys of about 3000 trees throughout University Park did not reveal others.

1949--Ten of the original 11 diseased trees were dead and an additional 600 trees were found exhibiting various stages of decline.

1950--Two hundred dead trees were noted and symptoms were observed on many others, extending the range about a half-mile in radius from the original location.

1951--Disease was found throughout University Park and in nearby College Park, Hyattsville and Riverdale. Approximately 400 dead or dying trees were counted.

Similar Symptoms, Due To Other Causes

Garren[2] recently reported what appeared to be a new disease of sweetgum in Alabama. His description, however, indicates that the disease differs materially from sweetgum blight in Maryland. He said, "This disease is definitely localized in the tree and does not develop throughout the perimeter of the crown as do most diebacks. It appears to be more prevalent and more destructive on younger trees, where it usually begins in the terminal portion of the leaders. Thus, 'leader dieback' is an appropriate descriptive name." He described internal symptoms associated with "leader dieback" as follows: "Inner bark and outer sapwood show streaks of blue-black to black discoloration extending downward beyond evident necrosis. Inner sapwood is normal, or with irregular, broken rings of light-tan discoloration."

Occasionally young sweetgums affected by blight in Maryland, especially in young, crowded, second-growth stands, show the first indication of the disease in the terminal portion of the leaders, just as in the leader dieback of sweetgum in Alabama and Mississippi. However, the sweetgum blight kills all parts of the tree rapidly, whereas the leader dieback normally is confined to the leader.

Sweetgums injured by construction work, changes of water level, gas leakage, girdling of the bark, or fire often manifest some of the same symptoms as sweetgum blight, but close examination usually will show differences. Fire, for instance, practically always chars the outer bark. In determining the presence of blight, it is advisable to avoid confusion by disregarding all trees affected by such factors.

An Appeal For Information

The sweetgum blight is very serious in University Park and vicinity. Research on the cause, means of spread, and other factors concerning the disease is under way.

Information is needed now on the dying of sweetgum in other parts of its range, either now or in the past. We solicit the cooperation of the various State and private agencies interested in sweetgums. An appeal is made to tree workers and observers to send in information on dying of sweetgums to their State plant pathologist at the Agricultural Experiment Stations and to the Division of Forest Pathology, U. S. Plant Industry Station, Beltsville, Maryland

DIVISION OF MYCOLOGY AND DISEASE SURVEY AND DIVISION OF FOREST PATHOLOGY, UNITED STATES DEPARTMENT OF AGRICULTURE

[2]Garren, Kenneth H. Leader dieback -- a "new" disease of sweetgum. Plant Dis. Reptr. 33 (5): 251-253. Sept. 15, 1949.

NEW OR UNUSUAL RECORDS ON PLANT DISEASE OCCURRENCE

THE GOLDEN NEMATODE IN PERU

By C. Bazán de Segura

The golden nematode of potatoes, Heterodera rostochiensis Wollenweber, was observed for the first time in Germany in 1923 and since that time has been considered indigenous to that country.

At the suggestion of Mr. A. L. Taylor of the Bureau of Plant Industry, Soils, and Agricultural Engineering, a search was initiated for this nematode in Peru, with the result that it was found at the end of February of the current year in potato roots sent from Tarma. This determination was confirmed by Dr. J. E. Wille, Chief of the Department of Entomology of the Centro Nacional de Investigacion y Experimentacion Agricola de la Molina, and by Mr. Taylor himself to whom we sent specimens of infested material.

At the moment a search is being made throughout Peru in order to determine the distribution of the pest. We are now of the opinion that Heterodera rostochiensis is indigenous in the Andes, where it possibly attacks other plants in addition to the potato, Solanum andigenum Juz. & Buk.

CENTRO NACIONAL DE INVESTIGATION Y EXPERIMENTACION AGRICOLA DE LA MOLINA, PERU, April 1952

ROOT-LESION NEMATODES PRESENT IN CHRISTMAS ROSE IMPORTED FROM HOLLAND

By C. G. Anderson and H. J. Jensen

Root lesion nematodes (Pratylenchus sp.) were discovered in the root systems of Christmas Rose (Helleborus niger) during a routine nursery inspection in December 1951. Most of the plants examined in two shipments from Holland were found to be infected.

The girdling and dieback of the root tips and the presence of local lesions commonly associated with these nematodes were observed on the roots of diseased plants. Numerous eggs, larvae, and adult root-lesion nematodes were recovered from the marginal region of healthy and necrotic areas. Living root-lesion nematodes were present in the root systems of infected plants after a four month storage period. A fungus, Cylindrocarpon sp., was usually isolated from the diseased plants but the primary damage was probably due to the nematodes.

OREGON STATE DEPARTMENT OF AGRICULTURE, SALEM; AND THE DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, OREGON AGRICULTURAL EXPERIMENT STATION, CORVALLIS

OOSPORES OF THE WHITE-RUST FUNGUS, ALBUGO CANDIDA, FOUND IN LEAVES OF HORSERADISH

By R. M. Takeshita, M. B. Linn and H. W. Anderson

The production of oospores of the white-rust fungus, Albugo candida (Pers.) Kuntze, on leaves of horseradish, Armoracia rusticana, has been obtained under greenhouse conditions in Illinois. As far as the writers are aware, oospore formation by this pathogen on horseradish has not previously been noted, although these spores have been reported in other crucifers. Kadow and Anderson[1] were unable to find oospores in old plant parts of horseradish. They stated that "so far as is known, oospores are not formed on this host and if formed are probably so rare as to be of little importance as a source of spring infections." Thus, the finding of oospores in the present instance suggests a mode of overwintering of the pathogen in addition to persistence of systemic mycelia in the crown area.

Oospores were observed during an investigation conducted in the winter of 1952 to determine the relative effectiveness of various methods of inoculation with conidia. Uniformly, severe infection was produced only in leaves maintained in a water suspension of conidia for 24 hours at 16-18°C. Approximately 14 days later, a grayish-black discoloration of the foliage, visible on both surfaces, began to appear on the plants so inoculated. A microscopic examination of such areas revealed an abundance of oospores which were identical in size and marking of the external wall with those of A. candida. Oospores were not found on plants inoculated by other methods.

[1]Kadow, K. J., and H. W. Anderson. 1940. A study of horseradish diseases and their control. Ill. Agr. Exp. Sta. Bul. 469: 531-583.

Limited experiments conducted to date indicate that temperature, intensity of infection, and the age of the infected leaf tissue may play an important role in oospore production.

Examinations were made of microscope slides bearing cross-sections of horseradish petioles infected with A. candida, and prepared by Dr. M. B. Linford of this Division from collections made in the field in October 1950. These revealed a few oogonia but no oospores. Since this material was collected in the field, it seems likely that oospores are also produced in commercial plantings. A search will be made this coming summer to determine the presence and relative abundance of oospores under natural conditions.

DIVISION OF PLANT PATHOLOGY, DEPARTMENT OF HORTICULTURE, UNIVERSITY OF ILLINOIS, URBANA, ILLINOIS

ROOT-ROT DISEASE-COMPLEX IN TOBACCO PLANT BEDS

By Wilbert A. Jenkins

With reference to a previous note (PDR 34(6): 177, 1950) attention of tobacco pathologists is again being called to a root-rot disease-complex in tobacco plant beds which is currently causing severe losses over a wide part of the Old Belt of Virginia and North Carolina. Numerous beds have been examined by the writer, while telephone calls and specimens received in the mail indicate that identical conditions are causing much concern to the growers in many areas in which the beds cannot be examined personally.

In every instance where microscopic examination of specimens has been possible, typical root mutilation characteristic of the feeding habits of nematodes of the meadow and allied groups is the outstanding symptom during the early phase of the disease. The tap root with its laterals typically shows severe mutilation, accompanied by lesions, blind rootlets, and root necrosis. On teasing fresh lesions apart, numerous stylet-bearing as well as non-stylet-bearing nematodes are found to be infesting the lesions. Later developments result in rapid soft rotting of the hypocotyl and complete severance of the root system from the remainder of the seedling at the soil level. By spreading the fingers like a rake and scooping into the infested areas of the plant beds, literally handfuls of plants are obtained. This is true because the seedlings have no attachment remaining to their respective roots. In areas where the soil is still moist and the air humidity is high, the leaves of such plants are still turgid and green and, except for noticeable stunting, the degree of injury would not readily be realized until the plant is touched and thus bowled-over. With reduction in soil moisture and humidity (as with removal of the plant bed cover) such seriously affected plants wilt and dry out very rapidly.

Although, as during the 1950 plant bed season, root-rot disease-complex of tobacco seedlings is not entirely confined to bed areas treated with calcium cyanamid, it is preponderantly so. It should also be noted that etiolation and yellowing of seedlings, so characteristic of the symptomatology during the 1950 season, has not been so prevalent during the current season. However, the season has thus far been characterized by periods of unusually high soil moisture, which doubtless accounts for the exceptionally rapid severance of the root systems through bacterial soft rot, to the exclusion of the yellowing and etiolation which are symptoms generally associated with less rapid root severance.

A survey of beds treated with soil fumigants revealed no root-rot disease-complexes, though it should be emphasized that the area and number of such beds are very small as compared with those treated otherwise.

VIRGINIA AGRICULTURAL EXPERIMENT STATION, CHATHAM, VIRGINIA

A CORRECTION

Our attention has been called to the following error in the article, "Internal Cork of Sweet Potatoes. Production of Corky Lesions in Core-Grafted Roots" by L. W. Nielsen. Plant Disease Reporter 36: 132-134, 1952.

On page 133, the last sentence in third paragraph reads . . . . "lesions observed in the roots may be related to . . . ." It should read, as it was stated in the manuscript "may not be related to . . . ."

"DISEASE IN PLANTS"

DISEASE IN PLANTS. An Introduction to Agricultural Phytopathology. By the late Neil E. Stevens and Russell B. Stevens. Waltham, Mass.: The Chronica Botanica Co.,; New York City: Stechert-Hafner, Inc. 1952. Price $4.75. Volume one of Chronica Botanica's "Manual of the Plant Sciences".

Because Dr. Neil E. Stevens was a former head of the Plant Disease Survey, this new text on plant pathology, started in collaboration with and completed by his son Russell B. Stevens, is naturally of special interest to us.

The junior author, in his preface, states that the book is the outcome of attempts to find a common ground for plant pathology courses to be given in two different Universities. "One fact was at once evident" he says. "It was not desirable to use the same illustrative material. Tennessee and Illinois are not far distant, but the major crops, the major interests of the students, are different. If this be true of two states which at one point are less than fifty miles apart, how much more evident must it be in states and countries more widely separated. Yet there must be some body of knowledge and a point of view which is common to plant pathology and plant disease problems everywhere."

The result of this search for a "common ground" is not really a text book at all, in the usual sense, but an interesting and provocative summation and discussion of the whole body of plant pathological knowledge, not omitting the unsolved problems. From it the reader experiences the same sense of exploration and enlightenment that characterized the senior author's conversation and writing throughout his career. It is good that his son seems to have inherited this original quality of mind.

The unique feature of this book is the unified concept of plant pathology that emerges from it. The plan of the book is partly responsible: it does not take up the study of individual diseases or of techniques, but leaves those subjects to the laboratory. Thus there results, not a series of separate details about plant diseases, but a logical and stimulating account of plant pathology as a whole. Moreover, more than any other book with which these reviewers are acquainted, this one succeeds in translating plant pathology into human terms. It starts out with a clear exposition of the relation of plant pathology to the world's food problems, and of plant pathologists to the rest of the world, and this universal viewpoint is maintained throughout.

It might be thought that such a book would be too general and remote to serve as the basis for a "practical" course in plant pathology. Nothing could be further from the truth. It is more and more evident that a worker in any field must not only be proficient in his individual tasks but must be able to evaluate his own special interests in relation to his science as a whole. In this sense this book is more "practical" by far than the conventional text. Quite apart from the grasp of relations and proportion that it imparts, however, it has an entirely sensible down-to-earth quality that makes it eminently "practical" in the usual meaning of concrete.

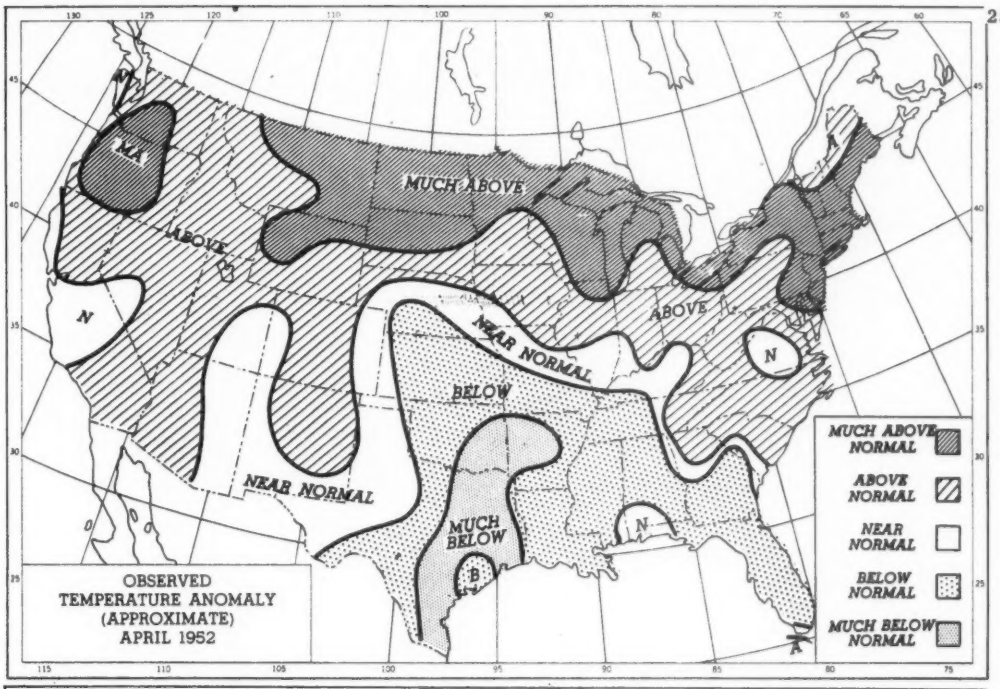
The chapter on "Economy of Disease Control" demonstrates this practicality. It discusses topics ranging from whether control is or is not advisable in particular instances and how much growers really are interested in controlling diseases, to the "quack" in plant pathology, finally coming back to "disease control and world economy." All of this puts control on a firmer foundation actually than any discussion of control measures as such could do.

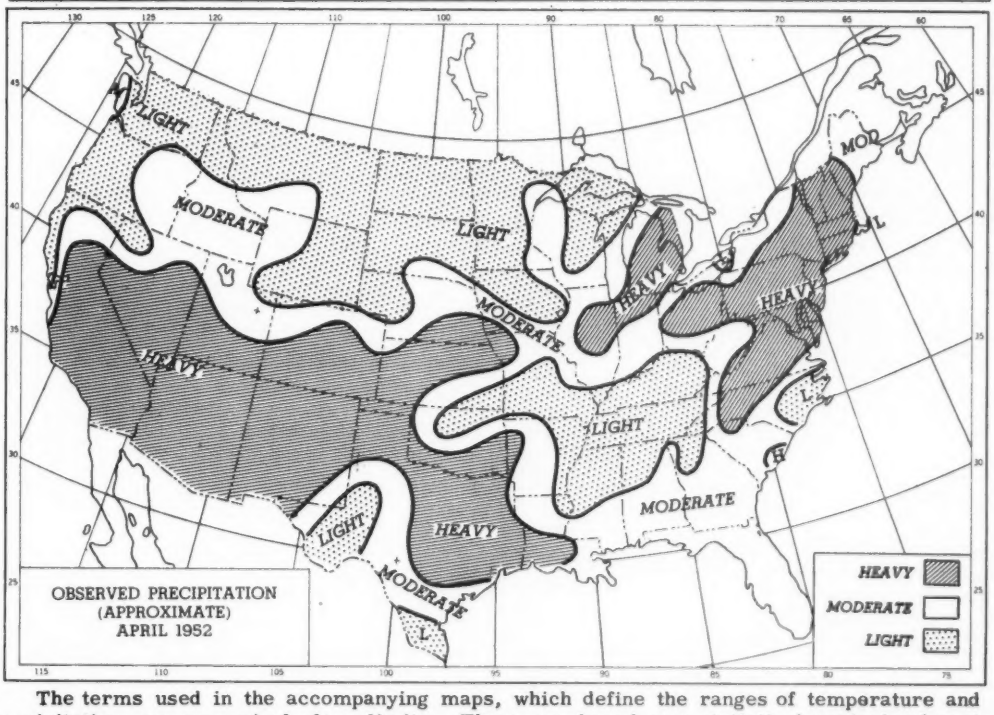
Again, the final chapter "Handling plant disease problems" takes the sensible and obvious, although not often expressly stated, viewpoint that the grower has an equal responsibility with the plant pathologist in meeting new problems. The "alert grower", the authors say, "will seek, promptly, the very best available professional advice"; he has "distinct obligations", among them is being perfectly frank about his local problem since he knows more about it than anyone else; besides this, in the interval before research can produce results he has "a direct obligation really to study the situation and see whether or not the circumstances point to possible control." They point out that many times successful control measures are evolved from farmer observation and experience long before research has found the cause of the trouble or explained why the empirical controls work. The plant pathologist, they say, contributes to the common problem a wider experience with disease in general, and "it is hoped" a more objective viewpoint.

Concerning newly discovered diseases, the authors have a word of warning: "The experience of the last three decades, in the United States at least, strongly suggests that from the purely practical point of view the safest course is to regard every new disease as potentially serious and probably infectious, and to act accordingly. The added cultural cost of such treatment will usually not be large. The saving may be very great."

This book seems to be remarkably free from errors. On page 22, however, the term "micron" mentioned several times in connection with virus particle size, should obviously have been "millimicron".

An unusually readable style is maintained throughout. The illustrations are well chosen; they include several facsimiles of historical interest. Sources of material are critically documented in every case and there is an extremely useful index. The volume is well-printed and attractively bound. -- P.R.M. and J.I.W., Plant Disease Survey





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.